Vol. LXXXIV OCTOBER, 1926 No. 4

ANNALS of SURGERY

A Monthly Review of Surgical Science and Practice

Edited by
LEWIS STEPHEN PILCHER, M.D., LL.D.
of New York
With the Association of
JAMES TAFT PILCHER, B.A., M.D.
and the Collaboration of
W. SAMPSON HANDLEY, M.S., M.D., F.R.C.S.
of London

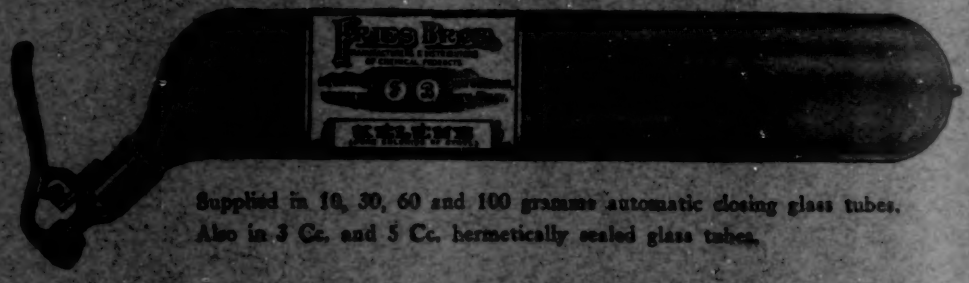
J. B. LIPPINCOTT COMPANY, PUBLISHERS
MONTREAL PHILADELPHIA LONDON

Entered at the Post Office at Philadelphia and admitted for transmission through the mails at second-class rates.
Price $10.00 a year.

KELENE

(Pure Chloride of Ethyl)

For Local and General
ANESTHESIA



Supplied in 10, 30, 60 and 100 grammes automatic closing glass tubes.
Also in 3 Cc. and 5 Cc. hermetically sealed glass tubes.

The automatic closing tubes require no valve
Simply press the lever

Widely used for many years

Sole Distributors for the U. S. A. and Canada
MERCK & CO.
St. Louis NEW YORK Montreal

PYRAMIDON

For the purpose of temporarily minimizing and relieving pain, pending operative procedures, a 5-grain tablet of Pyramidon proves an effective analgesic.

Pyramidon, thus employed, in no way interferes with subsequent procedures leading to anesthesia, general or local; nor does Pyramidon have any disturbing effect on the digestive tract or upon the organs generally.

H. A. METZ LABORATORIES, INC.
17 HUDSON ST. NEW YORK

ANNALS of SURGERY

VOL. LXXXIV OCTOBER, 1926 No. 4

ALKALOSIS*

BY FREDERICK A. BOTHE, M.D.

OF PHILADELPHIA, PA.

ALKALOSIS as a complication in surgical conditions has been studied very carefully by many observers during the past ten years. The clinical, pathological, and blood chemistry changes in these cases are more or less generally understood and the existence of a severe toxæmia has been recognized. However, most studies have been focussed on its occurrence in cases of pyloric, duodenal, or high intestinal obstruction, the result of a disturbance in the motor function of the upper gastro-intestinal tract. In this paper I wish briefly to discuss alkalosis and report six cases in which it developed following operation, five of which illustrate that alkalosis should be thought of as a post-operative complication in conditions in the abdomen other than the above-mentioned.

We can divide the surgical conditions in which alkalosis occurs into two groups, the pre-operative group and the post-operative group. The pre-operative cases should be subdivided into those of pyloric or duodenal obstruction and those of peptic ulcer which have developed alkalosis under an alkaline therapeutic regime. It is of importance to recognize it in cases of pyloric or duodenal obstruction before operation, because if immediate operation is not necessary, the medical treatment of this condition undoubtedly decreases the operative risk. It is possible also that treatment before operation in such cases may prevent its occurrence or lighten its severity, should it develop after operation. In 1923, Hardt and Rivers reported a study of 48 cases of duodenal ulcer treated by the Sippy method, some of which developed definite toxic symptoms associated with renal changes, increased blood urea, and normal or increased carbon dioxide combining power of the plasma. This they found was more prone to occur in cases with renal complications. In 1919, Harrop reported a case of bichloride poisoning in which the carbon dioxide combining power rose to 80 volumes per cent. and tetanic and convulsive seizures developed following two intravenous injections of sodium bicarbonate. These instances show that alkalies are contra-indicated in cases with renal damage.

The etiology of the severe toxæmia associated with the resulting alkalosis is not known. Ellis, in a study as to the cause of death in intestinal obstruction, isolated a toxin from the duodenal contents which when injected into lower animals reproduced the symptoms. He concluded that a toxin was

*Read before the Philadelphia Academy of Surgery, May 3, 1926.

elaborated in the cells of the greater part of the mucosa of the small intestine, chiefly in those of the duodenum. Whipple, Stone and Bernheim, using closed duodenal loops in dogs, showed also that a toxic substance was formed which when injected into animals reproduced the original picture. They feel that the toxic substance is formed either in the duodenal mucosa or is a product of bacterial autolysis or both. Hayden and Orr raise the question whether the toxæmia may not be due to the absorption of the products of protein destruction rather than to the primary substance responsible for the protein destruction. The great fall in blood chlorides and decrease of chlorides in the urine, together with the benefit derived by the intravenous injections of chlorides in these cases shows the demand the body has for chlorides. In either case it is very evident that the chlorides serve as a protective agent to the body in fighting off this condition.

Vomiting is quite persistent and may be in large or small amounts. Gastric lavage gives only temporary relief and affords an aid in differentiating a case of post-operative alkalosis from one of an atonic stomach with dilatation. Abdominal distention is usually, though not always, absent in alkalosis. As a result of the persistent vomiting and great fluid loss the patient becomes greatly dehydrated, the cheeks are flushed, the blood-pressure falls, and the hæmoglobin is quite high.

Nervous irritability is increased and frequently the patients have muscular twitchings and numbness and tingling of the extremities. In the more severe cases, the twitchings become more pronounced and the picture of tetany develops; in fact, it may go so far as spasmodic convulsive seizures which involve the entire body. Tetany usually develops later, but in some severe cases it develops early and may be the earliest sign.

The renal picture is usually quite characteristic. In 1923, Brown, Eusterman, Hartman and Rountree brought out the importance of the renal complications in this condition. The urine becomes scanty, kidney function is diminished, the urinary findings show evidence of marked damage, *i.e.*, albumin, casts, red and white corpuscles are present and the blood urea is elevated. If the kidney damage is severe enough the patients become drowsy, exhibit mental confusion and in the very severe cases the symptoms of uræmia develop. Tucker, in 1922, reported eight fatal cases from the Mayo Clinic in which uræmia developed following gastro-enterostomy.

The changes in the blood chemistry in these cases are quite constant, of great diagnostic value and in severe cases serve as a guide in the treatment. There is a decrease in the blood chlorides, a rise in the blood nitrogen, and normal or elevated CO_2 combining power. The fall in the blood chlorides is usually the first change noted and it is followed by the rise in the blood nitrogen and the CO_2 combining power of the blood plasma. The work of McCallum and his associates, and McCann, showed that the plasma chlorides fell and that the alkaline reserve was increased in pyloric closure in the lower animals. Tilletson and Comfort pointed out that the blood nitrogen was increased in cases of intestinal obstruction, while Hayden and Orr later

demonstrated all three changes in the blood chemistry in cases of pyloric and duodenal obstruction.

The cause of the fall in the plasma chlorides is not clear. Some investigators believe it is probably related to the increased protein destruction; others are inclined to attribute it to the loss of gastric juice by vomiting. Against the latter idea is the fact that some cases have very little vomiting but still the fall in plasma chlorides is very great. However, the loss of hydrochloric acid by vomiting probably plays some part, whether it be only secondary or is the primary cause.

The nitrogen retention in the blood may be due to either increased tissue destruction or renal injury. Tilletson and Comfort in their work feel that the increased tissue destruction is the primary cause and that decreased elimination and loss of fluid may be a factor. The report of Brown, Eusterman, Hartman and Rountree demonstrated rather conclusively that the renal injury was a big factor; and the urinary findings showed the kidney damage to be quite marked.

There is thought to be some relation between the elevation of the CO_2 combining power of the blood and the appearance of symptoms of tetany. McVicar reported a series of cases in which he observed that tetany developed most frequently in cases having a carbon dioxide combining power in the vicinity of 100 volumes per cent. However, it was not constant and one of his cases had a carbon dioxide combining power of 161 volumes per cent. with no clinical evidence of tetany. Doctor Jopson recently had a case of alkalosis following cholecystectomy, which had a CO_2 combining power of 110 volumes per cent., which showed no evidence of tetany. Other cases are reported with a low carbon dioxide combining power in which tetany developed. Hence, as Hayden and Orr pointed out, we must consider the possibility that tetany is primarily due to a disturbance in the protein and, secondarily to a disturbance in the inorganic salts.

In arriving at a diagnosis of alkalosis we cannot always depend on the clinical picture. Persistent vomiting, evidence of marked dehydration, diminished urinary output, and the presence of uræmic or tetanoid tendencies are the findings which would establish the diagnosis clinically. However, the changes in the blood chemistry are more constant and should be studied early in cases of persistent vomiting. Early diagnosis is of importance so that treatment may be instituted before too great renal damage has occurred.

In considering treatment the pre-operative and post-operative cases should be taken up separately. In the pre-operative group the lesion producing the stasis of the intestinal canal is of primary importance and the resulting alkalosis is secondary. If immediate surgical intervention is not necessary medical treatment is indicated. Repeated gastric lavage should be given to relieve the stasis, and normal saline and 10 per cent. glucose should be administered to combat the depletion of chlorides and the renal insufficiency. In the more serious cases the saline and glucose can be given intravenously. The changes in the blood chemistry and the improvement in the excretory

function of the kidneys aid one's judgment in determining the time of operation.

As a post-operative complication, alkalosis is more serious and its development and course is more rapid. In a small percentage of cases it does exist in a mild degree and repeated gastric lavage with the administration of saline and glucose, other than intravenously, clears up the alkalosis. However, in the severe cases, it has been found to be more efficient to give 500 c.c. of normal saline and 500 c.c. of 10 per cent. glucose intravenously two to three times daily, depending on the severity of the case. The glucose is given to help spare the protein and promote diuresis. Small doses of dilute hydrochloric acid and ammonium chloride have been given by mouth or rectum with beneficial results, but have not been used in the cases reported in this paper. Along with the intravenous therapy, daily estimations of the blood chemistry should be made as these findings furnish a better index of the patient's condition than does the clinical picture. If under medical treatment the vomiting persists, the blood chemistry shows no improvement, and the evidence of renal damage increases, jejunostomy is indicated. Following jejunostomy, gastric lavage and intravenous therapy are continued and the patient is fed through the jejunostomy tube. Some cases exhibit marked renal damage very early and in these instances, though jejunostomy be performed early, the prognosis is grave. As intravenous therapy may be given for a long time, it is well to preserve the superficial veins. If tetany develops calcium is indicated and is administered most efficiently intravenously in 5 c.c. doses of a 10 per cent. solution of calcium chloride. Alkalies are contra-indicated; this is mentioned because of the wide use of sodium bicarbonate in cases of persistent vomiting, whether it be given by mouth, proctoclysis or is used to lavage the stomach.

Case I.†—A boy fifteen years of age was admitted to the Presbyterian Hospital February 6, 1926, on the service of Doctor Speese, with a chief complaint of pain in the lower right quadrant of the abdomen. He had a typical attack of appendicitis eight days before admission, with generalized peritonitis and the formation of an appendiceal abscess. At operation an appendiceal abscess was found; the appendix could not be delivered so drainage was instituted. He vomited an ascaris lumbricoide the first day after operation. Forty-eight hours after operation he began to vomit several times a day in small amounts, which was not relieved by gastric lavage. The sixth day after operation the blood chemistry showed blood urea nitrogen 32, plasma chlorides 280, and the CO_2 combining power was 52 volumes per cent. The urine showed definite evidence of kidney damage and the urinary output fell. Glucose and saline were given intravenously twice daily and clinically there was some improvement for the next forty-eight hours. The blood urea nitrogen was 31, the plasma chlorides had risen to 400 and the CO_2 was 46 volumes per cent. The next day he became greatly distended and started to vomit again, so that a jejunostomy was performed. He was relieved by this procedure and the distention disappeared. A fecal fistula developed at the site of the original operation the following day. The urine still showed a cloud of albumin and many red blood cells. The intravenous therapy had been continued and four days after the jejunostomy a study

† The normal blood urea nitrogen is 12 to 18 mgs. per 100 c.c., the normal for the plasma chlorides is 560 to 650 mgs. per 100 c.c., the normal CO_2 combining power is 50 to 65 volumes per cent. The blood urea nitrogen and not the total blood urea has been studied in these cases.

of the blood chemistry showed the blood urea nitrogen was down to 8 and the plasma chlorides had risen to 514. The blood was lost and a CO_2 determination could not be made. The patient's general condition had improved and he was able to take liquids freely and some soft food by mouth. At this time the drainage from the fecal fistula became very profuse and the patient rapidly wasted from inanition. A transfusion was given and an attempt was made to close the jejunostomy but there was no healing power and the closure broke down. The patient sank rapidly and died forty-eight days after operation. Post-mortem examination showed many dense adhesions throughout the abdomen with a fecal fistula in the right iliac fossa around which there was a pocket of fecal material. There was also some fecal material walled off around the jejunum which had formed around the site of the jejunostomy. Both kidneys showed subacute toxic nephrosis with passive congestion.

This case showed marked renal damage early and illustrates the unfavorable prognosis in such cases. Perhaps jejunostomy may have aided more had it been performed earlier.

Case II.—The patient was a male sixty-eight years of age who had a cholecystectomy performed on December 5, 1925, by Doctor Speese. Forty-eight hours after operation hiccoughs, nausea and vomiting developed which persisted for eight days when it ceased. A small amount of solid food was taken and the same symptoms returned. On the eleventh day the patient's condition became more serious than at any time since operation; the pulse became elevated and the temperature rose to 100. The urinary output had decreased from forty-three ounces to twelve ounces in the last twenty-four hours. In view of the great dehydration and severity of the symptoms a jejunostomy was performed. Following jejunostomy the daily intake of fluids by mouth was gradually increased. On the fifteenth day solid food was taken by mouth and again there was a recurrence of the vomiting and hiccoughs. A diarrhœa also developed at this time with 5 to 7 movements a day. Lavage by a Jutte tube was given and saline was administered by hypodermoclysis to maintain the daily fluid intake. The urinary output again diminished at this time. Lavage was discontinued after five days and the patient went on to an uneventful recovery.

Clinically this case showed the picture of alkalosis; unfortunately, however, there were no estimations made of the blood chemistry to confirm it. An interesting and unexplained fact is that many examinations of the urine failed to reveal any evidence of renal damage which should be expected in such a severe case of alkalosis as this one. This case illustrates the fact that one should not hesitate in doing a jejunostomy in this group of cases, when they do not respond to medical treatment, before the kidney damage is too great. Jejunostomy is sometimes performed too late in cases in which it would have benefited had it been performed earlier and for this reason the operation has not gained the favor it should have.

Case III.—The patient was an adult male admitted to the Presbyterian Hospital, March 8, 1926, on the service of Doctor Hodge. He had a history of non-obstructive duodenal ulcer with a positive X-ray for which a posterior gastro-enterostomy was performed. Forty-eight hours after operation the patient began to vomit clear green fluid which at no time was foul. Seventy-two hours after operation the blood chemistry showed a blood urea nitrogen of 36, blood chlorides of 362 and a CO_2 combining power of 41 volumes per cent. Intravenous treatment of normal salt solution and 10 per cent. glucose was started. The vomiting improved greatly but forty-eight hours later the patient vomited several times in large amounts which was not aided by lavage. On the eighth day he was explored; the stomach was found to be pulled over to the right by

adhesions and adherent to the abdominal wall. There was no evidence of inflammation and neither the proximal nor distal loops were dilated. The proximal loop seemed quite long so an entero-anastomosis was performed. The patient was relieved for forty-eight hours when the vomiting recurred and was more marked than at any previous time. The urinary output was greatly diminished though fluids were given beneath the skin to keep up the fluid intake in view of the persistent vomiting. The urine at this time showed a trace of albumin and an occasional hyaline cast. The blood urea nitrogen was 24, the plasma chlorides were 328 and the CO_2 combining power had risen to 73 volumes per cent. The abdomen was not distended. The patient began to feel drowsy and slept in short naps. Intravenous therapy was started and in twenty-four hours the vomiting had ceased, the urinary output had increased to seventy-one ounces. In forty-eight hours the patient was much brighter, was not vomiting and could take small amounts of fluid by mouth with no discomfort. Examination of the blood chemistry at this time showed that the blood urea nitrogen had fallen to 15, the plasma chlorides had risen to 446 and the CO_2 combining power had fallen to 65 volumes per cent. The urine showed a trace of albumin, 75–100 white blood cells, 2–3 red blood cells, and an occasional hyaline cast. From this time on recovery was uneventful and the urine returned to normal.

After the gastro-enterostomy, while there was some alkalosis present, it seems that mechanical factors were the most important in the etiology of the vomiting. But after the entero-anastomosis the picture of alkalosis was more definite and we believe was the cause of his vomiting. This case was very interesting because it illustrated both mechanical factors and alkalosis as a cause of vomiting.

Case IV.—This patient was a male, forty-one years of age, admitted to the Presbyterian Hospital, February 9, 1926, on the service of Doctor Jopson, complaining of severe pain in the abdomen. About eighteen hours before admission the patient was seized with sudden severe abdominal cramps which gradually localized in the lower right quadrant. He vomited and his abdomen became distended. An enema and turpentine stupes were given to relieve the distention, with no effect. On admission the patient was suffering from considerable abdominal pain, the abdomen was greatly distended, there was marked rigidity and peristalsis was almost absent. The temperature was 98.8, pulse 104, and respirations were 24. Operation was decided upon in face of evidence of a general peritonitis because of sudden onset and rapid progress. A perforating lesion with outpouring of infectious material seemed quite possible. A right rectus incision revealed a generalized purulent peritonitis. The appendix was not acutely inflamed so further exploration was made. The exploration of the upper abdomen was negative except for free pus. However, on exploring the pelvis, a perforated necrotic diverticulum of the sigmoid was found. Drainage was instituted and the wound was partially closed. After operation the daily fluid intake was maintained by giving saline by hypodermoclysis and a Jutte tube was passed to control the vomiting. The patient's general condition was somewhat better forty-eight hours after operation and an occasional peristaltic wave could be heard. The Jutte tube was then removed. The third day after operation the patient became more toxic, the pulse became very rapid and weak. Examination of the chest revealed many coarse râles, the temperature rose to 101.4 and the urinary output fell. He then sank rapidly and died seventy-two hours after operation.

This case did not suggest alkalosis clinically but the blood was studied just before death to see what change would be found. The blood urea nitrogen was 37, the blood chlorides were 360 and the CO_2 combining power was 81 volumes per cent., showing that alkalosis had developed. This case shows the importance of having a study of the blood chemistry in cases with persistent vomiting. In this particular case no other therapeutic measures could

have been instituted, however, a knowledge of the blood chemistry may be very helpful in the treatment of a less fulminating case.

Case V.—A male patient forty-five years of age was admitted to the service of Doctor Speese at the Presbyterian Hospital in April 16, 1925. At operation a chronic appendicitis and multiple calcified mesenteric lymph-nodes were found. An appendectomy was performed and a lymph-node was removed for diagnosis. Forty-eight hours after operation the patient developed hiccoughs, nausea and vomiting for which gastric lavage was given. Blood chemistry studies showed the blood urea nitrogen had risen to 65, the plasma chlorides had fallen to 320 mgms. and the CO_2 combining power was 59 volumes per cent. Intravenous treatment of glucose and saline was administered twice daily and on the fourth day the blood urea nitrogen had fallen to 45, the plasma chlorides were 360 and the CO_2 combining power was 45 volumes per cent. The vomiting had ceased but the hiccoughs persisted intermittently. Examination of the urine at this time showed a trace of albumin with 35–40 white blood cells and 50–60 red blood cells to the high power field. Previous examinations of the urine had been essentially negative. A definite phlebitis, developed in the right saphenous vein, at this time. On the fifth day the patient suddenly became very weak, the pulse was rapid and feeble and death occurred in one-half hour. The death was quite suggestive of embolism but unfortunately a post-mortem examination could not be obtained.

The alkalosis which had developed in this case following operation was benefited by intravenous therapy. This was evidenced by the fact that the patient had improved clinically and the blood chemistry studies revealed changes toward the normal. It is reasonable to suppose that this case would have gone on to an uneventful recovery had not some other complication developed.

Case VI.—The sixth case has previously been reported in detail by Doctor Jopson in the issue of the Annals of Surgery in August, 1925. The patient was a male, thirty-seven years of age, who was operated upon for a large right scrotal hernia which was reduced with considerable difficulty because of the many coils of small intestine in the sac. A Stetton modification of the Bassini method of herniotomy was performed. The patient was nauseated and vomited the first day after operation. Gastric lavage was given but the vomiting continued so a Jutte tube was inserted. The material obtained from the stomach consisted of dark brown, granular appearing fluid. The abdomen was somewhat distended and hiccoughing occurred at short intervals. Normal saline was given by hypodermoclysis. The Jutte tube was removed when the fluid became clear, but the vomiting recurred, so the Jutte tube was reinserted. On the fourth day the patient was decidedly worse, became delirious and had convulsive seizures accompanied by cyanosis and unconsciousness. The hands were flexed at the wrists but the typical tetanoid position was not observed. The urinary findings in this case were normal, the blood urea nitrogen was 47, the CO_2 combining power was 88 volumes per cent. and the blood calcium was 12 mgms. per 100 c.c. Unfortunately an estimation of the blood chlorides was not made. Five c.c. of a 10 per cent. solution of calcium chloride was given intravenously for three days. It is of interest that oxygen inhalations which were given with the idea of combating the extreme cyanosis appeared to have a decided effect in checking the convulsions.

Doctor Jopson felt at the time he reported this case that perhaps it furnishes a warning against the too prolonged use of the Jutte tube in cases of acute dilatation of the stomach, intestinal obstruction and peritonitis. The clinical picture along with the findings in the blood chemistry are so typical that we may consider this case an example of post-operative alkalosis.

In summarizing, one may say that the number of cases reported in this

paper is not great enough to draw any definite conclusions. However, they do present enough evidence that in cases of persistent vomiting an early study of the blood chemistry should be made to see what changes have taken place. The benefit derived from the administration of chlorides and glucose in cases of pyloric and duodenal obstruction with the resulting toxæmia and alkalosis has already been shown. It is impossible to say how great a factor alkalosis is as a post-operative complication in other conditions in the abdomen which have persistent vomiting. In very fulminating cases, as the case presented which had a perforated diverticulum of the sigmoid, the treatment of the alkalosis would probably be of little if any value. However, early treatment of the alkalosis in a less fulminating case may not only benefit the patient, but in some instances be life saving.

In concluding I wish to extend my appreciation to Doctors Jopson, Hodge, and Speese for permission to study and report the above cases.

REFERENCES

[1] Brown, G. E., Eusterman, G. B., Hartman, H. R., and Rountree, L. G: Toxic Nephritis in Pyloric and Duodenal Obstruction: Renal Insufficiency Complicating Gastric Tetany. Arch. Int. Med., 1923, vol. xxxii, pp. 425–455.

[2] Ellis, J. W.: The Cause of Death in High Intestinal Obstruction. ANNALS OF SURGERY, 1922, vol. lxxv, pp. 429–448.

[3] Haden, R. L., and Orr, T. G.: Chemical Changes in the Blood After Intestinal Obstruction. Jour. Exper. Med., 1923, vol. xxxvii, pp. 365–375.

[4] Haden, R. L., and Orr, T. G.: The Chemical Changes in the Blood after Pyloric Obstruction. Jour. Exper. Med., 1923, vol. xxxvii, pp. 377–381.

[5] Haden, R. L., and Orr, T. G.: The Effect of Sodium Chloride on the Chemical Changes in the Blood of the Boy after Pyloric and Intestinal Obstruction. Jour. Exper. Med., 1923, vol. xxxviii, pp. 55–71.

[6] Haden, R. L., and Orr, T. G.: The Chemical Changes in the Blood of Man after Acute Intestinal Obstruction. Surg., Gyn. and Obst., 1923, vol. xxxvii, pp. 171–180.

[7] Hardt, L. L., and Rivers, A. B.: Toxic Manifestations Following the Alkaline Treatment of Peptic Ulcer. Arch. Int. Med., 1923, vol. xxxi, pp. 171–180.

[8] Harrop, G. A., Jr.: The Production of Tetany by the Intravenous Infusion of Sodium Bicarbonate. Bull. Johns Hopkins Hosp., 1919, vol. xxx, pp. 62–63.

[9] Jopson, J. H.: Acute Dilatation of the Stomach and Tetanoid Convulsions Following Operation for Hernia. ANNALS OF SURGERY, vol. lxxxii, pp. 303–304.

[10] MacCallum, W. G., Lintz, H., Vermilye, H. N., Leggett, T. H., and Boas, E.: The Effect of Pyloric Obstruction in Relation to Gastric Tetany. Bull. Johns Hopkins Hosp., 1920, vol. xxxi, pp. 1–7.

[11] McCann, W. S.: A Study of the Carbon Dioxide Combining Power of the Blood Plasma in Experimental Tetany. Jour. Biol. Chem., 1918, vol. xxxv, pp. 553–563.

[12] McVicar, C. S.: A Discussion of the Clinical and Laboratory Clinical Findings in Certain Cases of Obstruction in the Upper Gastro-intestinal Tract. Jour. of the Med. Sciences, 1925, vol. clxix, pp. 224–235.

[13] Tileston, W., and Comfort, C. W., Jr.: The Total Non-protein Nitrogen and the Urea of the Blood in Health and in Disease, as Estimated by Folin's Methods. Arch. Int. Med., 1914, vol. xiv, pp. 620–649.

[14] Tucker, W. G.: Uremia Following Gastro-enterostomy, Eight Cases. Wisconsin Med. Jour., 1922, vol. xx, pp. 528–529.

[15] Whipple, G. H., Stone, H. B., and Bernheim, B. M.: Intestinal Obstruction. The Defensive Mechanism of the Immunized Animal Against Duodenal Loop Poison. Jour. Exper. Med., 1914, vol. xix, pp. 144–165.

ERRORS IN DIAGNOSIS OF SURGICAL CONDITIONS

By Marie F. Gregory, M.D.
AND
Frances E. Vosburgh, M.D.
of New York, N. Y.
FROM THE FIRST SURGICAL (CORNELL DIVISION) NEW YORK HOSPITAL

During the years 1918 to 1925, the house surgeons on Doctor Gibson's service (the Cornell Division) at the New York Hospital, have made a weekly report of the cases; reporting the total number of operations, the infections, the deaths and the mistaken diagnoses. The following study has been made from these reports, covering a period of six and one-half years.

During this period, from January 1, 1918, to July 1, 1925, there were about 10,173 operations performed in this service at the New York Hospital. The total number of errors in diagnosis reported were 268, or 2.63 per cent. The errors were more frequent in women than in men, 56 per cent. of error in females, 29; 1 per cent. in males and 16.9 per cent. in which the sex was undetermined because the charts could not be found on account of inaccuracies in writing.

The study of mistaken diagnoses was undertaken, first by classifying all cases according to the pre-operative diagnosis, then by classifying all the post-operative diagnoses, to determine the frequency with which various conditions were either mistaken for some other, or were missed.

Both lists are headed by appendicitis, and gastro-intestinal conditions lead in frequency. Diseases of the female genital tract are second in importance, and those of the liver and gall-bladder third. Errors in the other systems are so scattered and infrequent that they hardly need special mention. Diseases of the pancreas appear five times on the list of missed diagnoses, and only once was pancreatic disease mistaken. This indicates that the pancreas is rather easily forgotten, and should be considered in the differential diagnosis of abdominal conditions.

The study of certain diseases or diagnoses was then taken up in detail, to find the frequency in which mistakes were made, and if possible, to suggest how they might have been avoided, in order to make a more accurate differential diagnosis.

The minimum and maximum percentage of errors in the six months' period were, respectively, .70 per cent. and 4.8 per cent.

The list of conditions in which mistaken diagnoses were made in three or more instances is as follows: 1. Acute appendix, 52. 2. Salpingitis, 29. 3. Gastric ulcer, 19. 4. Fibro-myoma uteri, 19. 5. Cholelithiasis, 17. 6. Cholecystitis, 17. 7. Ectopic gestation, 11. 8. Ovarian cyst, 10. 9. Adenitis, 10. 10. Chronic appendix, 10. 11. Intestinal obstruction, 9. 12. Inguinal hernia, 7. 13. Duodenal ulcer, 6. 14. Adhesions, 5. 15. Femoral hernia,

4. 16. Cysts, 4. 17. Fibroma, 3. 18. Carcinoma of gall-bladder, 3. 19. Peritonitis, 3.

TABLE I.

By systems these mistakes may be classified as follows:

Gastro-intestinal	Female pelvic	Liver and gall-bladder	Kidney	Spleen
Acute appendix...52	Salpingitis...20	Cholecystitis..17	Pyonephrosis.....2	Splenomegaly 2
Gastric ulcer.....19	Fibroids.....19	Cholelithiasis 17	Nephrolithiasis.....2	Carcinoma....1
Chronic appendix...10	Ectopic.....11	Carcinoma... 3	Perinephritic absc.....2	3(1%)
Intestinal obst..... 9	Ovarian cyst 10	Abscess of liver..... 1	6(2%)	
Ulcer..... 6	Retroversion 2	Ecchinococcus cyst..... 1		
Adhesions.. 5	Parametritis. 1	39(18%)		
Perforation. 2	Ca. of uterus. 1			
Ca. of rectum..... 2	Tub-ovarian absc..... 1			
Hemorrhoids 2	Bicornuate uterus.... 1			
Pyloric obst. 1	T. B. salpingitis..... 1			
Ca. of stomach..... 1	67(31%)			
109(48%)				

There were forty other conditions in which mistakes were demonstrated in one or two instances.

TABLE II

The following instances of missed diagnoses were detected: 1. Chronic appendicitis, 27. 2. Acute appendicitis, 24. 3. Ovarian cyst, 23. 4. Adhesions, 22. 5. Salpingitis, 19. 6. Undetermined, 16. 7. Cholecystitis, 14. 8. Cholelithiasis, 11. 9. Ectopic gestation, 11. 10. Inguinal hernia, 8. 11. Fibromyoma, 7. 12. Normal pregnancy, 6. 13. Peritonitis, 5. 14. Intestinal obstruction, 5. 15. Gastric carcinoma, 5. 16. Lymphosarcoma of intestines, 5. 17. Sarcoma, 4. 18. T. B. peritonitis, 3. 19. Carcinoma of bile passages, 3. 20. Carcinoma of intestines, 3. 21. Tubovarian abscess, 3. 22. Gastric ulcer, 2. 23. Chronic pancreatitis, 2. 24. Acute pancreatitis, 2. 25. Femoral hernia, 2. 26. Pyelitis, 2. 27. Volvulus, 2. 28. Carcinoma of uterus, 2. 29. Pelvic abscess, 2. 30. Endometritis, 2. 31. Cyst, 2. 32. Adenitis, 2. 33. Thyroglossal cyst, 2. 34. Carcinoma of breast, 2. Forty-nine other conditions presented only one case each.

TABLE III.

Classified by systems, these missed cases may be tabulated as follows:

Gastro-intestinal	Female pelvic	Liver and gall-bladder	Kidney	Pancreas
Chronic appendix.....27	Ovarian cyst...23	Cholecystitis..14	Pyelitis..2	Chronic pancreatitis.....2
Acute appendix.....24	Salpingitis.....19	Cholelithiasis 11	Abscess..1	Acute pancreatitis.2
Adhesions....22	Ectopic gestation.....11	Carcinoma... 3	Perin....1	Carcinoma.1
Ulcer..... 8	Fibromyoma.. 7	Ecchinococcic cyst..... 1	Ectopic kid. ...1	5(2%
Carcinoma.... 8	Normal pregnancy..... 6	29(14%)	5(2%)	
Obstruction... 5	Carcinoma.... 4			
Lymphosarcoma..... 3	Abscess..... 3			
Volvulus..... 2	Endometritis.. 2			
Perforation... 1	75(35%)			
Foreign body.. 1				
101(47%)				

Discussion of the most important diagnoses, with details of the conditions with which they are confused: 1. Appendicitis. 2. Gall-bladder disease. 3. Salpingitis. 4. Ovarian cysts. 5. Pregnancy, normal and extra-uterine.

In sixty-two instances cases diagnosed as appendicitis were demonstrated at operation to be: Salpingitis, 9. Ovarian cyst, 9. Gall-bladder disease, 8. Perforated ulcer, 6. Ectopic gestation, 5. Intestinal obstruction, 4. Adhes-

ions, 3. Undetermined, 3. Fibroma uteri, 2. T. B. peritonitis, 2. T. B. intestines, 1. Pyelitis, 1. Peritonitis, 1. Diverticulitis, 1. Nephritic abscess, 1. Ectopic kidney, 1. Vaginitis, 1. Perinephritic abscess, 1. Mesenteric thrombosis, 1. Pneumonia, 1. Foreign body, 1.

In 39 instances that were otherwise pre-operatively diagnosed as: Gall-bladder disease, 12. Salpingitis, 9. Ulcer, 6. Ectopic gestation, 3. Intestinal obstruction, 2. Parametritis, 1. Ovarian cyst, 1. Abscess of rectus sheath, 1. Fibroma, 1. Adhesions, 1. Hernia, 1. Carcinoma of cæcum, 1. Appendicitis was found to be present at operation.

Appendicitis.—Appendicitis heads the list for frequency of mistaken and missed diagnoses. Acute and chronic cases are grouped together. In mistaken diagnoses pelvic conditions are in the lead, with 25 of the 62 cases showing disease of the uterus or adnexa.* This makes the frequency of mistakes much greater in women than in men. The gall-bladder was the offending organ in 8 cases, and some part of the gastro-intestinal tract in 19 cases. The kidney was involved in 4 instances.

This seems to show that the diagnosis of appendicitis is a little too freely made. More careful pre-operative study might give more definite localization and a more accurate diagnosis. However, since the diagnosis of appendicitis usually means operation, and real pathological conditions are found on exploration and remedied.

Of the 39 cases where appendicitis was found on operation, nearly one-third were diagnosed as gall-bladder disease—either cholecystitis or cholelithiasis. This emphasizes the difficulty in localizing abdominal pain in some cases.

Salpingitis was the pre-operative diagnosis in 9 cases. It is very likely that there was some doubt in the minds of the surgeons in these cases, for if the diagnosis had been absolutely certain, no operation would have been done in the acute cases, of which there are a few in this number.

Ulcer of the stomach or intestines was suspected in 6 cases, ectopic gestation in 3, intestinal obstruction in 2, and the other 7 cases were scattered in diagnosis.

Gall-bladder Disease.—Among the cases diagnosed as gall-bladder disease, there were 3 cases where a diagnosis of cholecystitis was made and cholelithiasis found and 1 diagnosed as inflammatory disease proved to be carcinoma. These are not actual mistakes when all gall-bladder conditions are considered together. This leaves 26 cases where trouble with the gall-bladder was suspected but not found. Appendicitis was found in 17 cases, diseases of the pancreas in 3, of the stomach or duodenum in 3 and the other 3 were liver abscess, torsion of omental fat and 1 of undetermined pathology.

* It is not always advisable to try to make too fine a diagnosis between acute salpingitis and appendicitis. A number of cases were operated on notwithstanding that the weight of evidence pointed toward a salpingitis, as we feel that it is much safer, if there is any doubt, to operate as we do not wish to run the risk of neglecting to remove an inflamed appendix with a possible high mortality.

In 10 cases diagnosed as appendicitis, gall-bladder disease was found, also 4 cases of perforated ulcer, 2 of adhesions and 1 each of ecchinococcus cyst, fibroma, and salpingitis.

Salpingitis.—Salpingitis, acute and chronic, were grouped together, and 22 cases were found in which this diagnosis was incorrect. Acute or chronic appendicitis (equally divided) was found in 8 cases. Ectopic gestation was the next most frequent error, there being 5 cases, and ovarian cyst third with 2 cases. Adhesions (1 case complicated by pregnancy) were present in 2 cases. One case each of endometritis, cholelithiasis—T. B. peritonitis and carcinoma of the bladder were found.

Cases in which the diagnosis of salpingitis was missed numbered 18, and the pre-operative diagnoses were not widely scattered. Appendicitis was suspected in one-half the cases, extra-uterine pregnancy and fibromyoma in 3 cases each, overian cyst in 2 and gastric ulcer in 1.

Ovarian Cyst.—Ovarian cysts were diagnosed in 8 cases where they were not found. The conditions found were pregnancy 3 cases (one extra-uterine and one with adhesions), T. B. salpingitis, acute appendicitis, salpingitis, retroperitoneal cyst, and fibromyoma, of each, one case.

The diagnosis of ovarian cyst was missed 22 times. The pre-operative diagnoses being appendicitis 9 cases, salpingitis 5, extra-uterine pregnancy 2, fibromyoma 3, and 1 each for cholecystitis, gastric ulcer and carcinoma of the uterus. This is one instance when more careful history or physical examination should have reduced the number of errors. It was the only case where the number of missed diagnoses greatly exceeded the number of mistaken diagnoses.

Ectopic Gestation and Pregnancy.—The diagnosis of extra-uterine pregnancy was made in 11 cases which proved on operation to be other conditions. Two of these were normal pregnancies, one complicated by fibromyoma uteri. Two ovarian cysts were found, and the rest of the series were inflammatory conditions of the adnexa or the appendix, equally divided.

Cases in which pregnancy, either normal or extra-uterine, were not diagnosed but were found were more numerous. In this series the pre-operative diagnosis was of an inflammatory condition of the appendix or adnexa in three-fourths of the cases. In two normal pregnancy was found, in ten there was extra-uterine pregnancy. Two cases were diagnosed as fibromyoma where normal pregnancies were found, while one ectopic was thought to be an ovarian cyst and one a subinvolution of the uterus.

It is not always feasible to employ all the refinements of diagnosis, when a patient has a palpable mass in the pelvis with symptoms, it is generally a sufficient warrant for operation.

The eleven mistaken diagnoses recorded under extra-uterine pregnancy where another condition is found, represent particularly the view that with any suspicion of an extra-uterine pregnancy, immediate operation should be performed. Some of these conditions would probably have been more accurately defined if more thorough examination and prolonged observation had seemed wise.

THE SURGICAL ASPECT OF BLOOD DYSCRASIAS ASSOCIATED WITH SPLENOMEGALY*

By John Speese, M.D.
of Philadelphia, Pa.

Any discussion of blood dyscrasias associated with splenomegaly must take into consideration a review of the function of the spleen and its action in health or disease on the blood and the blood making organs. Without going into the subject of splenic enlargements in general, I shall discuss certain forms of splenomegalies which are accompanied by disease of the blood, and which are becoming of increasing importance because of the attempt to cure or arrest the diseased process by removal of the spleen. I shall review briefly the principal facts brought out by the intensive investigations which have been carried out in this field of research and those facts proven by clinical experience.

It is an accepted fact that the spleen plays a definite rôle in the formation of red blood cells in embryonic life but this function is lost either shortly before or after birth, but is resumed in certain anæmic states in which the spleen reverting to the embryonic type, produces red cells. During infancy and in early adult life, when the germinal centres of the spleen are most active, lymphocytes are found and gain entrance to the circulation by contractions of the spleen. Other varieties of leucocytes, such as the polymorphonuclear and myelocytes, do not occur normally in the spleen, although they may appear in certain forms of anæmia and in myelogenic leukemia.

A more important function of the spleen is that which is concerned in the destruction of worn out red cells. These cells are taken up in the spleen by phagocytes which aid in their fragmentation, then carried to the liver or elsewhere in the reticulo-endothelial system for final destruction and the hæmoglobin changed into bilirubin. The function of red blood cell destruction is carried on after splenectomy by the bone-marrow or lymph-nodes which undergo hypertrophy and assume the splenic function of red cell destruction. In this manner is explained the fact that removal of the spleen is not followed by any alteration in the formation of bile pigment nor is there any accumulation of pigment in the circulation as the red blood cells are phagocytosed anywhere in the body and carried to the liver. When red cell destruction is so pronounced that the hæmoglobin is more than the normal amount which can be taken care of by the liver and transformed into bilirubin, the excess of pigment accumulates in the blood and jaundice occurs.

The rôle which the spleen plays in digestion is not known, although some inter-relationship between it and the stomach has been supposed to exist from the earliest times. Inlow's [1] experiments on this subject and a critical review of the literature have led him to conclude that a definite pepsinogenic function

* Annual Oration before the Philadelphia Academy of Surgery, April 5, 1926.

of the spleen has not been demonstrated and that the relation of the spleen to gastric secretion is probably vascular, the diminution in the amount of gastric juice secreted after splenectomy being attributed to decreased gastric blood supply from injury to the gastro-splenic circulation.

Other observers have thought that the spleen may have an internal secretion which functionally influences some portion of the digestive apparatus by way of the blood stream or of activating one or more of the digestive enzymes by means of this secretion. Krumbhaar [2] believes that a specific splenic hormone is activated by passage through the liver. Mayo on the other hand asserts that the spleen does not possess an internal secretion of importance, not only because removal of the normal organ does not disturb metabolism, but also because of its extremely limited sympathetic nerve supply, as the organs concerned in internal secretion act through the sympathetic nervous system.

On purely hypothetical grounds, Kahn [3] thinks it probable that the spleen develops certain enzymes which are important to its function but it is equally evident that the function of the spleen is shared by other lymphoid or adenoid tissues of the body and that on removal of the organ, its function is continued by these other collaborating structures. After splenectomy the nodes along the greater curvature of the stomach and omentum hypertrophy and become red and new ones develop in the region of the extirpated spleen. Hyperplasia of the lymphatics develops, first in the vicinity of the portal vessels and then inside the liver lobes. This compensatory hyperplasia explains the liver enlargement common after removal of the spleen.

The spleen has been regarded as an important structure in filtering from the circulation various microörganisms and thus acts as a defensive mechanism against disease. In all probability its function as a filter has been overestimated. That it plays a rôle in resisting infection seems to be proven from the vast amount of experimental evidence which indicates that the spleen assumes an active part in the development of resistance against various acute infections, and this function after splenectomy may be assumed vicariously by other body structures.

The effects of splenectomy have been studied extensively in animals, and also in man in those cases in which the organ has been removed for rupture. We should bear in mind, however, that these observations have been noted in healthy individuals, and far different ones may be observed when the spleen is removed for definite pathological processes. In the normal splenectomized animal a secondary anæmia develops either immediately or after a short interval and persists for two or three months. Sometimes the whole reaction is delayed according to Kettle,[4] and occasionally the blood count becomes higher than before splenectomy. There is an immediate increase in the number of leucocytes in which the polymorphonuclears may increase three or four times their normal number, the count falling to normal in three to four months. The increase in the resistance of the red blood cells to hæmolysis after splenectomy is probably due to certain changes in the red cells themselves. This resistance rapidly follows removal of the spleen and lasts for months

and even years. There is a lessened tendency to jaundice which Pearce [2] explains as being due to diminution in the concentration of the products of red cell disintegration as they reach the liver. After removal of the spleen, red cell fragmentation is carried on largely by the bone-marrow located at such a distance from the liver, that the products reach the liver so diluted that jaundice is unlikely.

Following splenectomy there is an increase in the excretion of iron, a decreased output of vital fat in the feces and an increase in the fat and cholesterin in the blood. The output of uric acid and urobilin in the urine and feces is diminished. The thyroid may enlarge after the spleen is removed, which supports somewhat the view that there is some inter-relationship between the two organs. It has been shown in animals that the red cells and hæmoglobin decrease, and regeneration of red cells is retarded by thyroidectomy. The administration of thyroid gland in normal animals may increase the red cells up to 15 per cent. These experiments Mackenzie [6] points out, show under controlled conditions that the thyroid hormone exerts a certain influence on hæmatopoiesis, and that in hypothyroidism the blood picture often suggests a severe anæmia, resembling either chlorosis or primary anæmia without proclaiming itself by the usual signs and symptoms of myxœdema.

The connection which exists between the spleen and the bone-marrow has been investigated extensively by Pierce [2] and his associates who state that the divergent results obtained are characteristic of all phases of experimental work on the spleen and doubtless are to be explained by the fact that removing the spleen takes away only one organ of a system composed of liver, spleen, lymph-nodes and bone-marrow, and that the interrelations of this system may cause compensations of great importance in determining the degree of blood distribution or regeneration and hence the degree of change in the bone-marrow. The relation, therefore, is rather a matter of changes which take place in the storage and utilization of the iron of the body than a specific hormone action. Bone-marrow becomes red after splenectomy because it begins to take on the function of storing iron, for there is such a great deficiency of the iron content of the blood that red cells cannot be produced, as a high iron content is necessary for this function.

That the spleen has some stimulating influence on the blood-forming organs is shown by the fact that splenectomized animals recover from the anæmia produced by hemorrhage or poisons less readily than do animals with a similar degree of anæmia in which the spleen is intact. Stradomsky claims for the spleen a two-fold hormone action on the bone-marrow, an inhibiting one on the over-production of red cells in the narrow, and a second one controlling under-production of the cells. Normally these two influences balance each other, but when the splenic hormone is abnormal or lacking, the bone-marrow produces unlimited quantities of red cells of inferior quality which die off rapidly and this increased erythrocytosis sets up an automatic vicious circle. The findings indicate that the bone-marrow may function to excess after having been released from the controlling influence of the splenic hor-

mone. After splenectomy, the administration of splenic extract is liable to give results confirming this rôle of the spleen as a regulator of blood destruction.

A more detailed description of the investigations on splenic function can be found in the admirable reviews of Pool and Stillman;[6] and Pearce, Krumbhaar and Frazier.[2]

Pernicious Anæmia.—In endeavoring to find some means of checking the blood destruction in primary or pernicious anæmia, splenectomy was suggested by those who thought the cause of the anæmia was to be found in the spleen. We are still in the dark concerning the cause of this disease, and the most that can be admitted at present is that the anæmia is the reaction of the body and particularly the blood system to some toxin of unknown origin which greatly affects the bone marrow, causing on the one hand stimulation and over-production at a time when the marrow is attempting to overcome the action of the toxin.

Kahn and Torrey[7] have studied thirty-three cases of anæmia, in all of which the Welch bacillus was found in the duodenum, associated with symptoms characteristic of pernicious anæmia. On the administration of hydrochloric acid the organisms disappeared and the blood picture improved rapidly. The significance of these results must be determined by future observations. However, the subsequent events in the development of pernicious anæmia, as described by Moynihan,[8] include so marked an effort of the bone-marrow to meet the demand made upon it that the parent cells of the erythrocyte are liberated before their offspring daughter and granddaughter cells are created. The greater the demand made upon the bone-marrow the earlier is the type of cell liberated and set adrift in the circulation, and the character of the nucleated cell thus found may afford an index to the gravity of the disease. Death ensues because of the persistence of the increased destruction of red cells at a time when the efforts of the medulla to form blood break down under the ceaseless strain imposed upon it.

I shall not discuss the pathology and symptomatology of pernicious anæmia in any detail. The general degenerative changes and blood picture are too well known to warrant repetition. The spleen itself is but slightly enlarged in most cases, a factor of some importance in considering its removal in the treatment of the condition. The bone-marrow is red and contains numerous nucleated cells and some hyperplasia of the myeloid tissue is present.

Attention may be called to the insidious character of the onset of pernicious anæmia, beginning with a slow and gradual loss of strength and the development of pallor. Other important clinical manifestations are an achlorhydria, which is almost constant, and if not present, seriously jeopardizes the diagnosis of pernicious anæmia. The nutrition of the patient generally remains good, although loss of weight may occur when glossitis develops with a resulting disinclination to eat. In no other form of anæmia is glossitis so common, and in some cases it is an early and most annoying symptom, and occasionally it is the first symptom complained of and the one for which relief is sought. Spinal cord degeneration in both the lateral and posterior columns may occur

fairly early in the disease and produce tingling and numbness of the extremities, or more severe neuritic pains and symptoms resembling tabes.

As infection undoubtedly plays a rôle in the etiology of pernicious anæmia it is necessary first in treating the disease to remove any possible foci of infection, whether arising in the mouth, gastro-intestinal tract, genito-urinary tract or elsewhere. For the anæmia itself the various therapeutic agents used to stimulate red cell formation may be tried, and some transitory benefit may be secured. Sooner or later transfusion must be employed. The use of small transfusions, frequently repeated, seems to be the method of choice, and so far as I have been able to judge in a large number of patients so treated, the results are equally good with citrated or whole blood. The benefit thus secured is due to stimulation of the bone-marrow with an increased production of red cells. By transfusion the patient may be brought to a " remission stage," remain fairly comfortable for many months, but hæmolysis soon reoccurs and the improvement secured by transfusion is lost and the patient lapses into a stage of profound anæmia. For these apparently hopeless cases, Walterhofer and Schramm [9] have improvised a new method of treatment in which the marrow cavity of the long bones was irrigated through small holes drilled in the cortex. The improvement which followed was so striking and occurred so soon after the irrigation that the authors were led to believe that it could not be a mere coincidence and are hopeful that the method may prove to be of permanent value in the treatment of pernicious anæmia.

Splenectomy has been advocated by many investigators in an attempt to cure pernicious anæmia and a large number of spleens have been removed in the past twelve years with results at first believed to be gratifying and hopeful. Some of the more recent accounts of this operation, however, are not so encouraging and many surgeons have given up splenectomy. The cases which seem to be most favorable are those in which hæmolysis is most active, with symptoms less characteristic of the disease. The prospect of benefit is better in early than in late cases, and only temporary relief of symptoms can be expected when the process has produced such changes in the bone-marrow that its power of cell reproduction is lost.

In spite of splenectomy Carslow [10] believes that the condition goes on to a final result no more satisfactory than that obtained by medical measures, and he condemns splenectomy as unsatisfactory in pernicious anæmia, especially if the spleen is small. It is possible he concedes, that there are several types of pernicious anæmia and that operation may prove satisfactory in one and not in another variety.

There is no doubt that splenectomy was overdone for many years and Krumbhaar [11] thinks the operation now is being neglected and not employed as frequently as it should be. He has collected 208 cases of splenectomy done in patients suffering from pernicious anæmia, there were 35 deaths within one month, the post-operative mortality was 16.8 per cent., 26 were unimproved and 144 improved.

The study of end-results at the Mayo Clinic shows that the results of

splenectomy were far better than anticipated at a time when the operation was discontinued. It was found that 21.3 per cent. of the patients survived the operation three years or more, living two and a half times as long as the average in a similar group of non-splenectomized patients at the same stage of the disease, and that 10.6 per cent. are alive after more than five years.

These results clearly indicate in at least one-third of the cases that the average life of patients with pernicious anæmia is greatly prolonged, and in about 10 per cent. the prolongation is sufficient to lead to the hope that cures may result in some cases. While transfusion gives temporary benefit, the improvement following splenectomy has been far greater than that obtained by transfusion or any other palliative method of treatment. The changes brought about by splenectomy are manifested by improvement in the condition of the blood and amelioration of the nervous symptoms although the nerve structure itself is unchanged. The progression of degenerative changes in the cord are delayed to some extent and the relapses, so commonly seen in other methods of treatment, become less severe and less frequent. The achlorhydria persists even after splenectomy, and the glossitis seems to be but slightly benefited. These various manifestations, Mayo states, indicate that the same agent which destroys the bone-marrow, which injures the spinal cord, which causes achlorhydria and glossitis, also affects the spleen, and that by removing the latter a vicious circle is interrupted.

Hæmolytic Jaundice.—A very important blood dyscrasia, the result of splenomegaly is that of hæmolytic jaundice. The cause of this condition is unknown but as Elliott and Kanavel [12] pointed out the enlarged spleen is the active agent in the destruction of the erythrocytes and in the production of the resulting anæmia. The jaundice is of the non-obstructive type in that we find bile in the stools and not in the urine. This disease, seen most frequently in the second and third decades of life, occurs in two forms, the congenital or Chauffard-Minkowski type and the acquired or Hayem and Widal type.

The congenital form often affects several members of the same family, does not cause such severe symptoms and the patients are often more icteric than sick. The life expectancy is greater and the patients sometimes live to the fifth or sixth decades. The acquired type is more serious, and usually begins with severe symptoms, the anæmia becomes grave and the course is more rapid. This type is more frequently a disease of the adolescent period.

The chief characteristics of hæmolytic jaundice are splenic enlargement, usually moderate; icteric tinge of the skin and sclera, absence of bile in the urine and the presence of bile in the stools. One of the outstanding diagnostic findings is the increased fragility of the red blood cells as is shown in the fragility test in which their resistance to hypotonic salt solution is determined. In the congenital type we find increased fragility of the red blood cells in several members of the family, which Giffin asserts may be an aid in establishing the diagnosis. The increase of the urobilin in the duodenal contents and the presence of urobilinogen in the urine are probably due to the increased

blood destruction. We find also the greatest number of reticulated red blood cells in this condition.

In the course of the disease the patients have definite crises, during which there is an elevation of temperature, chills, increase in size of the spleen, increase in the jaundice and general malaise. The crisis may last from a few to a number of days when the symptoms subside. However, the jaundice, while appreciably diminishing, does not completely disappear. In the acquired type the crises are usually more severe.

It is not uncommon for patients with hæmolytic jaundice also to have gall-stones, which producing an obstructive type of jaundice, makes the diagnosis more difficult. Giffin [22] reports that 58 per cent. of the cases operated on at the Mayo Clinic for hæmolytic jaundice had gall-stones, and Moynihan found that 60 per cent. of his cases were so affected.

The pathological changes in the spleen are by no means characteristic and resemble the changes found in splenic anæmia. The capsule and trabeculæ are thickened, the Malpighian bodies are few in number and atrophic; there is a pronounced fibrosis throughout with atrophy of the splenic pulp. Increase in blood pigment as determined by the presence of hæmosiderin, is not marked.

The results of splenectomy in this condition are most striking and gratifying. The jaundice diminishes in the first twenty-four hours after operation, and in a few days the skin often has a normal color for the first time in many years. The anæmia rapidly disappears, the patient's health is improved and they remain well, showing that the spleen was the active agent in the destruction of the red cells. The fragility of the red cell decreases but does not return to normal. At the Mayo Clinic [13] fifty-one splenectomies have been performed for hæmolytic jaundice with only three deaths. Temporary relapse several months after operation has been noted by Giffin, while Elliott and Kanavel report a case in which a crisis occurred before the patient left the hospital; both patients, however, ultimately recovered and have remained free of symptoms.

Banti's Disease.—Banti's disease and splenic anæmia are now regarded as one disease, although there are some observers who believe that the symptoms of Banti's disease are the result of a terminal stage of splenic anæmia.

Three stages of splenic anæmia are described: (1) A preascitic stage, in which splenic enlargement is present with or without anæmia, the patient undergoing gradually increasing weakness. (2) The transitional stage in which the most prominent symptom is diarrhœa; the anæmia and blood changes are pronounced; the liver is somewhat enlarged and jaundice may occur; (3) The ascitic stage or Banti's disease proper.

The disease is characterized by a clinical course in which there is a progressive increase in the severity of the symptoms, this stage may last for many years. Probably the first symptom noted is enlargement of the spleen, which at first is gradual, then rapid in the late stage of the disease when the spleen may reach huge proportions, although not becoming so large as the splenomegaly seen in Gaucher's disease.

The anæmia met with is of the secondary type, but may become more severe in those cases in which frequent hemorrhages from the gastro-intestinal tract are met with.

The cause of the disease is unknown, although its cure by splenectomy naturally has led many to suppose that the pathogenesis of the affection is to be found as a primary disease of the spleen itself. However, no one has succeeded thus far in demonstrating any single factor which could be regarded as the cause of splenic anæmia.

The chief pathological condition found in the spleen in splenic anæmia is a generalized fibrosis, compression atrophy of the Malpighian corpuscles and endophlebitis. Chaney [14] from the pathological and clinical study of sixty-nine cases states that no changes were found in the splenic tissue that would enable the pathologist to positively diagnose splenic anæmia, yet the abnormality was as characteristic of this disease as in others producing splenomegaly. The average weight of the spleen was found to be 1015 grams and the average age thirty-three years. The disease affected both sexes equally with apparently no familial tendency.

It is believed by some that splenic anæmia is a clinical entity and that fibrotic splenomegaly produces anæmia, irrespective of the initial cause of the splenic enlargement. Mayo [15] favors this view and believes that a patient with chronic splenomegaly who presents characteristics of chronic anæmia, but who is not relieved by treatment is probably a sufferer from splenic anæmia, and will probably be cured by splenectomy without regard to the cause of the disease. In support of this theory we have the clinical evidence of improvement or cures following splenectomy in intractable cases of splenomegaly with anæmia, occurring in syphilis and chronic malaria.

While ascites may be present in splenic anæmia without fibrosis of the liver existing, it is possible that improvement following splenectomy may be due partially to diversion of some of the blood going to the liver, thus relieving it of overwork or toxins from the spleen, which in time may result in those fibrotic changes which lead on to portal obstruction, ascites, the formation of varicosities, rupture of which may cause severe and serious hemorrhages from the œsophagus and stomach. These hemorrhages, so common in splenic anæmia, and the cause of the more advanced degrees of anæmia, may cease entirely after splenectomy in early cases. In cases in which the hemorrhagic tendency has existed for a long time splenectomy may benefit but not stop the bleeding. In some instances the hemorrhages are probably the result of toxins formed in the liver which produce erosion of the gastric mucosa, and in this type of case, relief is less apt to occur after the spleen is removed.

The treatment of splenic anæmia is essentially surgical and consists in splenectomy. Preliminary to operation medical treatment may be tried to improve the general condition of the patient, although but little can be hoped for from these measures. Transfusion, both before and after operation, particularly if blood has been lost from hemorrhages from the mucous membranes, is beneficial but has only a temporary effect on the anæmia. It is highly

important to operate in the earlier stages of the disease, before dense adhesions form between the spleen and surrounding parts, particularly the diaphragm; before pronounced anæmia, liver fibrosis and ascites develop. Unfortunately the early manifestations of splenic anæmia are not sufficiently characteristic to make an early diagnosis likely and the surgeon too often is asked to remove a spleen in the terminal stages, at a time when the risk is great and the mortality correspondingly high. The adhesions between spleen and diaphragm may be so dense that removal of the spleen is impossible on account of the danger of hemorrhage, and the surgeon in such cases may be compelled to abandon the idea of splenectomy. The mortality in the early cases is not over 10 per cent., whereas, in the late stages of the disease it is about 25 per cent.

While the mortality is lower in the earlier manifestations of the disease, many cases recover after splenectomy performed in the terminal stages with ascites and liver cirrhosis present. With such secondary degenerative changes existing, cure of the disease cannot occur, whereas permanent relief can be said to have been obtained in the majority of the operations done in the early stages. It should be remembered, however, that not a few of the patients have recurrence of symptoms even after the lapse of many years.

Krumbhaar's [11] compilation is the most comprehensive collection of the results obtained by splenectomy in splenic anæmia. Of 239 cases collected, the post-operative mortality was 13.6 per cent., if the cases are subdivided into those occurring in recent years, a reduction to 11 per cent. is obtained, and it seems entirely likely that even this comparatively low mortality will be lessened with an increasingly improved technic and a more proper selection of cases. This is demonstrated by Mayo's [15] report of 10 per cent. of deaths in 82 splenectomies for splenic anæmia. Most of the deaths occurred in patients operated on in a late stage of the disease, in which there was a high degree of anæmia, ascites, and cardiorenal degeneration.

Gaucher's Disease.—This disease, first described by Gaucher in 1882, and regarded by him as a malignant process, has received much attention from the pathological standpoint, but less consideration has been given to it clinically because of its infrequency.

We are indebted to Brill, Mandlebaum and Libman for much of our earlier knowledge of the disease. The recent study of Cushing and Stout [16] who have analyzed forty-four cases, serves to bring more forcibly to our attention many of the clinical manifestations of the disease. They found that the affection ordinarily appears in childhood and runs a more acute course than in adults, in whom it is more chronic in nature. Females are more commonly affected than males (67 per cent.). Splenic enlargement which is noted in all cases, is progressive until almost the entire abdomen is occupied by the smooth splenic tumor. The bronze or yellowish discoloration of the skin, face, neck and hands is one of the striking features of the disease. The eyes show a peculiar wedge-shaped thickening of the conjunctiva which is present in most cases. No marked disturbance in general health may be noted for a long period when a secondary anæmia may appear and the patient

develops a tendency to bleed from the mucous membranes or into the skin. The blood picture shows a consistent leukopenia, the red cells may be decreased to a marked extent in the cases which bleed freely. The disease has a family tendency. Ascites and jaundice are rare, although enlargement of the liver is noted in 73 per cent. of the cases. Pain over the spleen is often complained of and pain in the region of the long bones may be a warning symptom of bone destruction, caused by the action of the Gaucher cells on the bone-marrow, and leading on to fracture.

Gaucher's disease, pathologically, is restricted to those cases in which the characteristic large vesicular cells with small eccentric nuclei, are found engorging the sinuses of the spleen, lymph-nodes and bone-marrow, or are crowded about the liver lobules.

In general the treatment of Gaucher's disease by internal medication, transfusion or radiotherapy has met with slight transitory improvement of the patient or no success at all. Splenectomy has been done in twenty-nine cases, with six deaths, an operative mortality of 20 per cent., of twenty-three patients who survived the operation, sixteen have gained in weight and strength and the hemorrhages have ceased. The operation seems to have lengthened the life of these patients to some extent. It can be concluded that splenectomy so far, is the only method of treatment that has met with any measure of success, although it cannot be stated positively that this operation actually cures the disease; rather it acts by producing an amelioration of the symptoms.

Purpura Hemorrhagica.—Many theories have been advanced in an attempt to explain the nature of this disease. While disturbance in liver function has been held responsible by some observers, the theory of infection advocated by Giffin and Halloway [17] has received the support of most writers on the subject. The disease is usually secondary to localized infection which produces chemical toxins whose action on the endothelium of the blood-vessels probably is the cause of the hemorrhages. The marked reduction in the number of blood platelets, without apparent cause, which is the main feature of the disease led Frank to designate the affection as essential thrombopenia and Eppinger to call it thrombocytopenia.

Brill and Rosenthal [18] regard purpura hemorrhagica as a distinct clinical entity, the main features of which are as follows: Reduction in the number of blood platelets from a normal of two or four hundred thousand to one hundred thousand or lower. A few cases are on record in which the platelets could not be demonstrated and I have treated one case in which two competent hæmatologists were unable to find blood platelets. As a general rule when the platelet count falls below sixty thousand the hemorrhagic tendency of the affection begins to appear, and these manifestations become pronounced when the platelets are under ten thousand in number.

Careful blood examination is of the utmost importance in the study of purpura hemorrhagica. The coagulation time of venous blood is preserved but the bleeding time is prolonged from a normal of one to three minutes to ten minutes or even hours. The capillary resistance test is an important diag-

nostic feature of the disease and may be demonstrated by applying a tourniquet to the arm sufficiently tight to prevent the return of blood without obliterating the pulse. If a purpuric tendency is present the test is followed by the appearance of multiple petechiæ. Another important blood phenomenon is failure of clot to undergo retraction, even when a fair number of platelets are present in the blood. This observation is useful as a differential test in hæmophilia in which the clotting time may be greatly delayed but when the clot is formed retraction always occurs.

From the clinical point of view purpura hemorrhagica pursues either an acute or chronic course. The acute cases result in a quick recovery, run a short and fatal course or become chronic. In the acute fulminating type the patients are so prostrated and so desperately ill that operative interference is out of the question and transfusion seems to be of little or no benefit. In the chronic form remissions sometimes occur making the diagnosis difficult if the patient is seen during this period. The capillary resistance test may prove of value as an aid in diagnosis at this stage.

When the disease occurs as a chronic affection it is encountered most frequently early in life and particularly in girls. Hemorrhages occur from the mucous membranes, are seen in the skin as multiple petechiæ and appear intermittently, as a continuous oozing or there may be excessive loss of blood following slight degrees of traumatism. Occasionally hemorrhages from the mucosa of the bowel may cause infiltration of the intestinal wall producing abdominal symptoms of pain, rigidity, tenderness, nausea and vomiting, fever, and leucocytosis, all symptoms suggestive of appendicitis. If such symptoms occur before other and more pronounced manifestations of purpura hemorrhagica have been noted there is scarcely any way of avoiding an unnecessary operation unless by some circumstance the surgeon happily should think of using the capillary resistence test.

Little can be expected from the treatment of purpura hemorrhagica by measures non-operative in nature. There is no doubt in some of the less severe forms of the disease that transfusion, preferably by whole blood, is followed by cure and particularly if any underlying infectious process can be removed at the same time. In the case quoted above with complete absence of platelets the bleeding ceased after a single transfusion the platelets increased rapidly in number and the patient has remained well for several years. Such a result must be exceptional for the condition of other patients has been uninfluenced by transfusion and in occasional cases indeed the condition seemed worse after transfusion.

Although the spleen is but slightly enlarged in purpura hemorrhagica there is no doubt that it is to be held responsible for the destruction of platelets. Accordingly its removal has been advocated as a cure for the disease, and in the cases in which this operation has been performed the blood platelets increase rapidly in number and the hemorrhages cease almost immediately. In some cases the platelet increase reaches its maximum only after many weeks, but as a rule the count returns to a low level shortly after the splenec-

tomy. Clopton [19] has reported instances in which there is a tendency toward recurrence of the disease in a greatly modified form in which the hemorrhages are readily controlled.

The immediate cessation of symptoms and apparent cure of the disease after splenectomy show that this mode of treatment offers the best chance of cure. Clopton has tabulated forty-five cases in which twenty-seven are reported as cured, fifteen improved, one unimproved and two operative deaths. The operative mortality is surprisingly low in view of the grave condition of most of these patients at the time of operation.

There are other obscure splenic blood disorders in which further study is necessary in order to properly classify them, and which are so little understood that operative interference by splenectomy is ill-advised. Farley [20] and others have called attention to the difficulty in distinguishing between aplastic anæmia, purpura hemorrhagica and acute myelogenous leukemia. In the light of our present knowledge we must agree with the contention of Hanrahan [21] and most writers on the subject that splenectomy is contraindicated in lymphoid leukemia, polycythemia, and the rapidly progressive fulminating forms of hæmolytic jaundice, splenic anæmia and pernicious anæmia. Providing the spleen has been treated previously by radium, which reduces its size and also at the same time, reduces the number of leucocytes, splenectomy offers the best chance of cure in myelogenous leukemia. While the number of cases so treated is comparatively small, the results seem to justify the operation in carefully selected cases, properly treated by radium before splenectomy is undertaken.

BIBLIOGRAPHY

[1] Inlow: Amer. Jr. Med. Sci., 1921, vol. clxii, p. 325.
[2] Pearce, R. M., Krumbhaar, E. B., and Frazier, C. H.: The Spleen and Anæmia, Philadelphia, 1918 (Lippincott).
[3] Kahn: Amer. Jr. Med. Sci., 1923, vol. clxxxv, p. 214.
[4] Kettle: Brit. Med. Jr., 1922, December 23, p. 1207.
[5] MacKenzie: J. Am. Med. Assn., 1926, vol. lxxxvi, p. 462.
[6] Pool and Stillman: Surgery of the Spleen, 1923, Appleton and Co.
[7] Kahn and Torrey: Penna. Med. Jr., 1926, vol. xxix, p. 394.
[8] Moynihan: The Spleen and Some of Its Diseases, Philadelphia, 1921 (Saunders).
[9] Walterhofer and Schramm: Archiv. fur Klinische Chirurgie, 1923, vol. cliv, p. 407.
[10] Carslow: Brit. Med. Jr., 1922, December 23, p. 1204.
[11] Krumbhaar: Amer. Jr. Med. Sci., 1923, vol. clxvi, p. 329.
[12] Elliott and Kanavel: Surg., Gynec. and Obstet., 1915, vol. xxi, p. 21.
[13] Mayo, W. J.: Jr. Amer. Med. Assn., 1924, vol. lxxxiii, p. 815.
[14] Chaney: Amer. Jr. Med. Sci., 1923, vol. clxv, p. 856.
[15] Mayo, W. J.: Jr. Amer. Med. Assn., 1925, vol. lxxxi, p. 487.
[16] Cushing and Stout: Arch. of Surg., 1926, vol. xii, p. 539.
[17] Giffin and Halloway: Medical Clinics of N. A., 1923, vol. vii, p. 241.
[18] Brill and Rosenthal: Arch. of Int. Med., 1923, vol. xxxii, p. 937.
[19] Clopton: Annals of Surgery, 1925, vol. lxxxii, p. 413.
[20] Farley: Amer. Jr. Med. Sci., 1925, vol. clxx, p. 10.
[21] Hanrahan: Annals of Surgery, 1925, vol. lxxxi, p. 906.
[22] Giffin: Surg., Gynec. and Obstet., 1917, vol. xxv, p. 152.

FIBROSARCOMATOUS TUMORS OF THE SKIN OF THE TRUNK

CHARACTERIZED BY ATTENUATED DERMAL SURFACES

By Arthur E. Hertzler, M.D.
of Halstead, Kans.

This designation may be applied to a group of tumors clinically characteristic, but too often not recognized. The importance of early recognition lies in the fact that if a complete removal of the tumor is instituted, together with the capsule, a permanent cure is assured. If shelled out from this capsule a recurrence is most certain to take place which may be impossible of complete removal. They are fibrosarcomatous in structure, tend to grow slowly and metastasize by way of the lymphatics. In appearance and clinical disposition they resemble closely the fibrosarcomas sometimes seen in the popliteal space which the old English writers called "recurrent fibroids." These tumors differ from most sarcomas by their long duration. In this they resemble some of the slowly growing melanoblastomas. One of my cases existed for twenty-five years before rapid growth began.

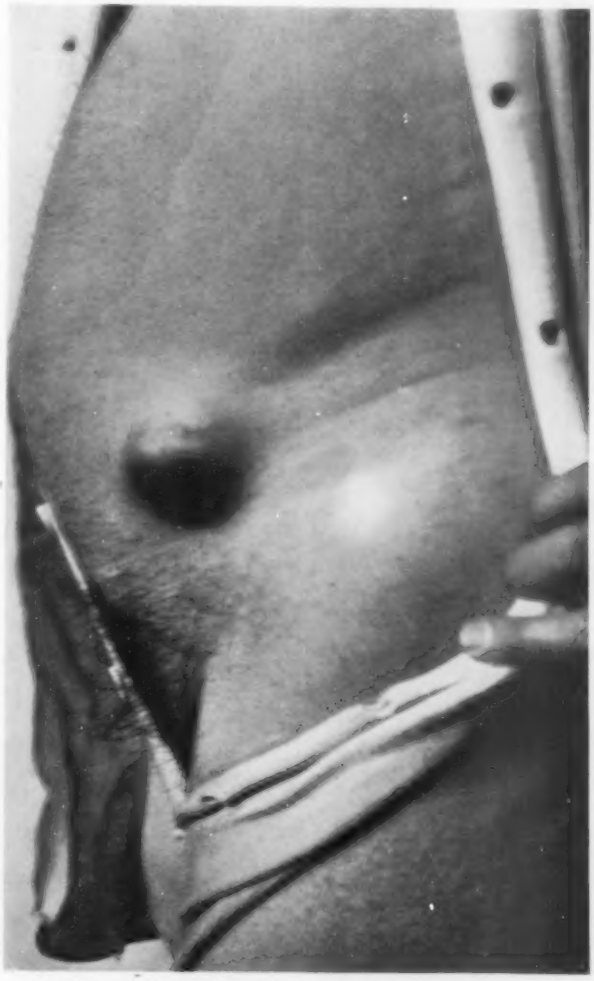


Fig. 1.—Tumor of the groin. The dark area represents the reddened surface. The sharp line of demarcation between this area and the normal skin is apparent at the upper part of the tumor.

Clinical Appearance.—The striking clinical feature is that the summit is covered with an attenuated skin of reddish or pinkish color (Figs. 1 and 2) resembling the covering of a spina bifida. In one of my patients the tumor was mistaken for an angioma

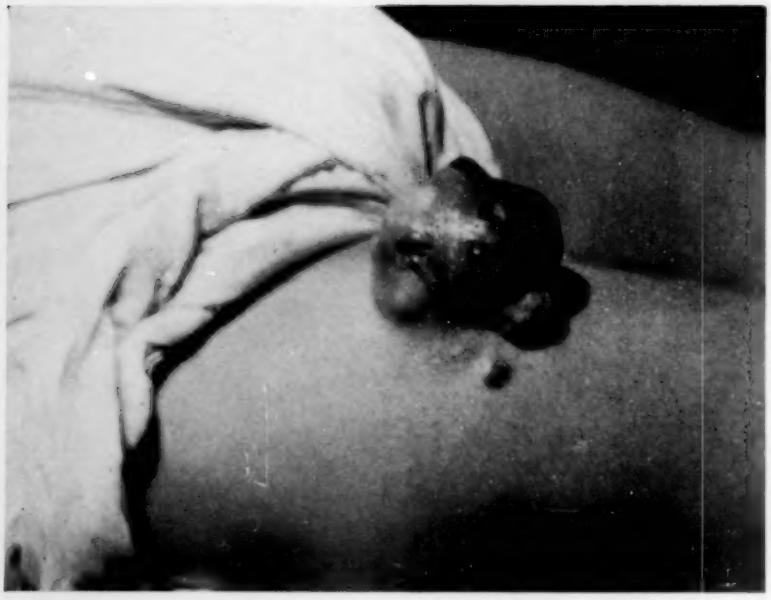


FIG. 3.—Tumor on the front of the thigh. The recent growth of the tumor is marked by the secondary bossiations and a nodule at some distance from the main tumor. The growth of the tumor has markedly extended the red area of the surface.

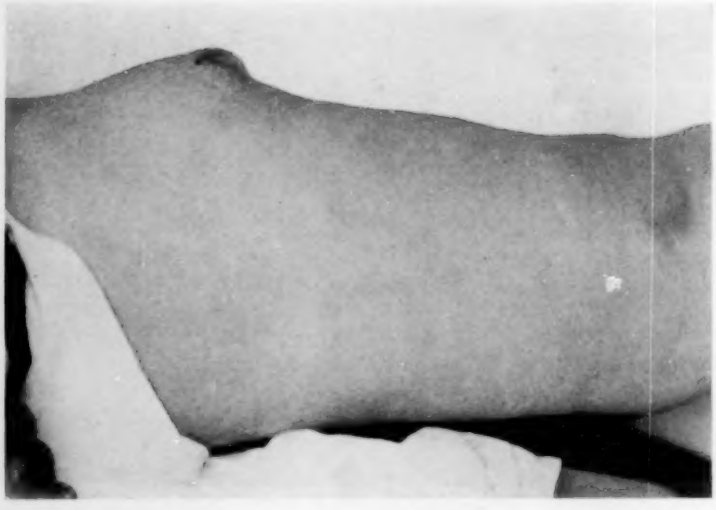


FIG. 2.—Tumor of the thigh. The red area is seen to occupy the surface of the tumor.

and treatment by galvanic needles caused it to grow rapidly. In some instances the line of demarcation is sharp as if an ulcerating mass were protruding (Fig. 3). When these tumors begin to grow rapidly, particularly when the tumor represents a recurrence, there is an actual protrusion of the tumor mass through the surrounding skin (Fig. 4). Sometimes hemorrhage takes place in the substance of the tumor causing a sudden enlargement. If the hemorrhage is near the summit ulceration usually soon follows. The primary tumor is spheroidal but recurrences may be bossilated (Fig. 5).

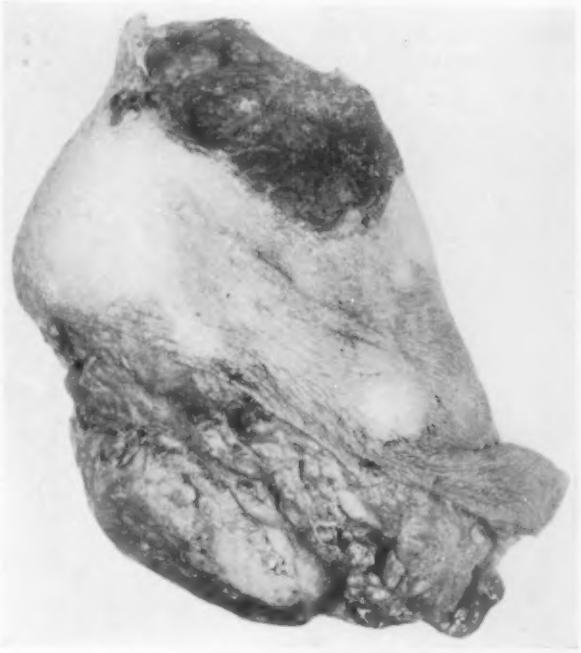


FIG. 4.—Tumor from over Poupart's ligament. The skin over the summit has been destroyed by the growth of the tumor beneath.

Incidence.—These tumors cannot be so rare for I have had twenty-two of them in the past twenty-five years covering an experience of some 5000 tumor cases. They are usually situated on the trunk, particularly about the groins and buttocks. I have seen only one above the clavicle and one just below the clavicle, two on the chest, one on the shoulder, four were located on the upper thigh and one on the calf. The remainder were located in the inguinal regions and about the buttocks.

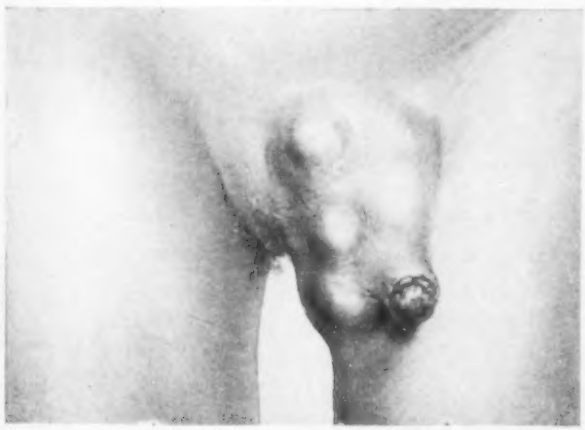


FIG. 5.—Recurrent tumor of the pubic region. Numerous bossilations are noted. At the apex the skin is destroyed by hemorrhage within the tumor.

Pathology.—These tumors present a complete encapsulation except over the surface where the skin is attached to the tumor. They glide over the deep fascia unless they have begun to invade the surrounding tissue. They are firm, dense, elastic on palpation. The cut surface shows wavy bundles more

or less parallel with each other (Fig. 6). The surface is pearly or pinkish and glistening. The slide shows that the skin epithelium is not thinned, as one would suspect from the reddish color of the surface. This appearance is due to the invasion of the skin by the tumor. The cells of the tumors are spindle form in character, arranged in more or less parallel bundles (Fig. 7). In the slowly growing tumors the connective tissue may predominate and most of the cells are small and spindle form, embedded in heavy bundles of fibrous tissue (Fig. 8). In some instances the cells are larger and may be distinctly endothelial in character. In one of my patients who had a tumor at the right costal margin the cell arrangement was distinctly that of an endothelioma. In recurrent tumors (Fig. 9), or those which have been irritated by treatment (Fig. 10), the cells may be a little broader and occupy the larger part of the field at the expense of the connective tissue, and the blood-vessels a little more in evidence. When these tumors begin to develop rapidly they invade the surrounding connective tissue. They first spread into the skin surrounding the summit and later become attached to the surrounding tissue. The areas of invasion and in the metastatic nodules they retain their fibrotic character. When they metastasize they do so by way of the lymphatics.

FIG. 6.—Cross-section of the tumor shown in Fig. 5. The tumor is made up of fibrous whorl-like bundles with more pinkish homogeneous areas between.

Symptomatology and Diagnosis.—The peculiar relation of the skin to the surface of these tumors makes the diagnosis evident at a glance. The relation they bear to the skin is rarely imitated by wens, but in these the skin covering the surface is rarely discolored. When the skin is so discolored, the imitation is perfect (Fig. 11). The wen is less firm and of course incision into the tumor makes the differentiation obvious. Lipomas occasionally attach themselves to the skin but in these the skin is not changed.

Sometimes large flat lipomas may protrude through the skin by a teat-like process. The protruding part may resemble the tumors in question very

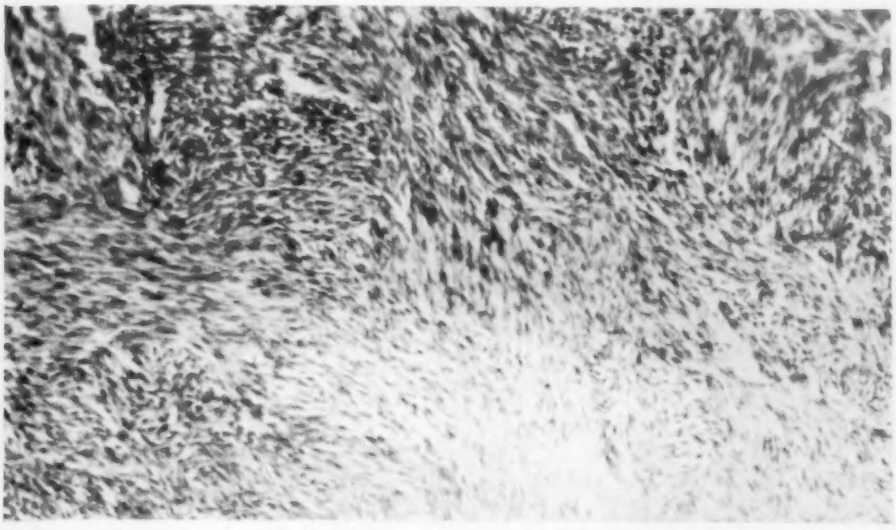


FIG. 7.—Slide from a primary tumor undergoing rapid growth. The spindle cells are arranged in irregular bundles and whorls.

closely while the bulk of the tumor is a typical lipoma (Fig. 12). Possibly these indicate a generic relationship between the two types of tumors. Gummas of the back may be attached to the skin and discoloration may pre-

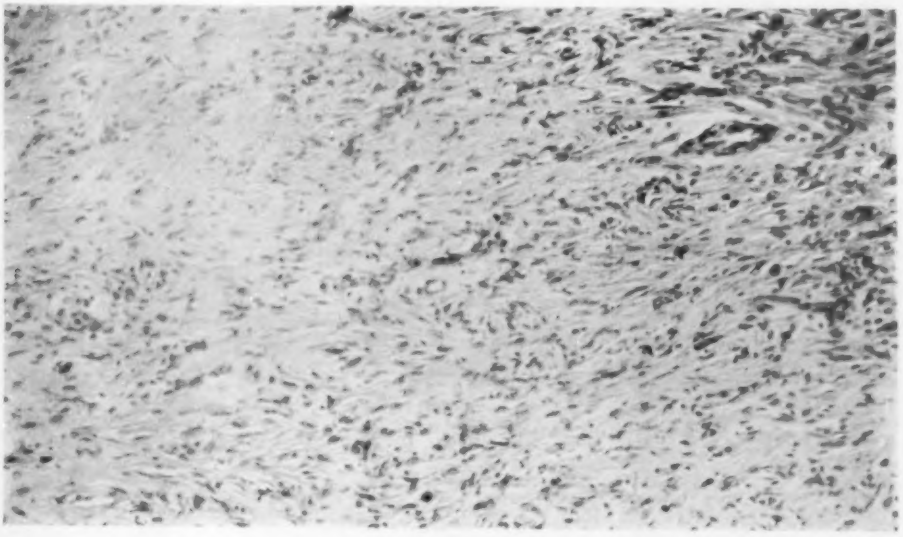


FIG. 8.—Slide from a quiescent tumor of many years duration. Spindle cells are scattered between heavy fibrous bundles.

cede rupture, but they lack the encapsulation and the duration is shorter. Because of this reddish surface they have been mistaken for angiomas. Con-

sidering the difference in density of the two tumors the differentiation is easy.

Treatment.—Complete excision of the tumor, together with its capsule

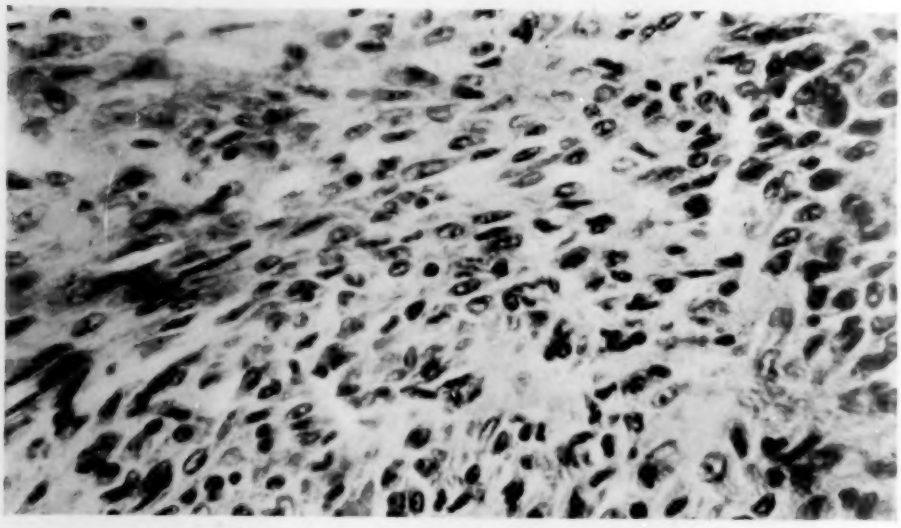


FIG. 9.—Slide from a recurrent tumor. The spindle cells dominate the field. Scattered among them are larger ovoid cells containing a more abundant protoplasm, a large nucleus and a prominent nucleolus.

fairly into the normal tissue about, results in a permanent cure. Even if recurrence follows inadequate excision (Fig. 5), a wide reoperation may still secure a cure. If the recurrent tumor is technically operable an attempt

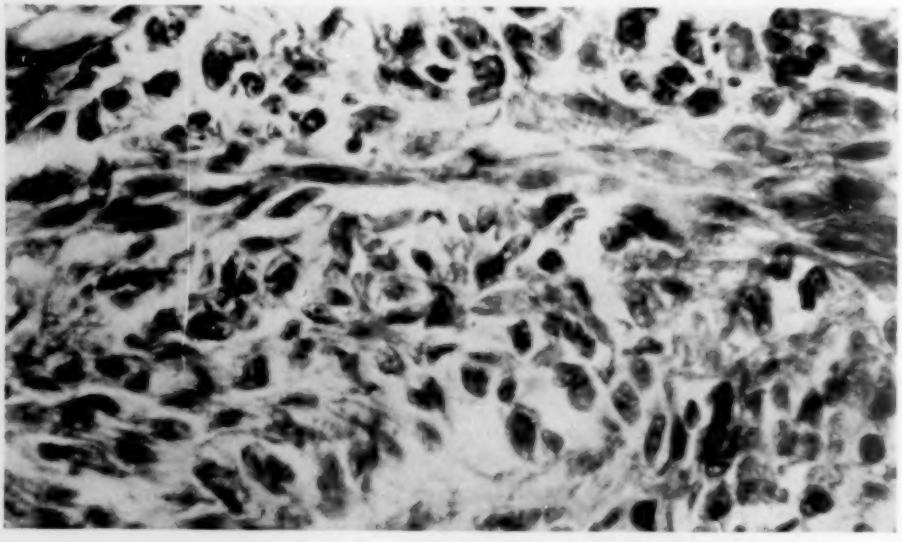


FIG. 10.—Slide from a rapidly growing tumor. Transitions between the spindle cells characteristic of the quiescent tumor and the ovoid cells of the recurrent tumors can be made out.

should be made. Even when there is regional metastasis the glands may be successfully removed. I operated on one patient seven times, during my

apprenticeship and the patient has remained well now twenty years. Operated on in a proper manner the first time it is not necessary to remove the neighboring lymph-glands.

Comment.—There seems to be nothing in the literature describing these tumors. Being so intimately attached to the skin it is fair to assume that they arise from some tissue belonging to this structure. They resemble so closely certain tumors derived from melanin-containing cells, that a close relation to these tumors seems likely. (Hertzler and Gibson, Melanoblastomas of the Foot. ANNALS OF SURGERY, 1914, vol. xxxvii, p. 89, July.) This possibility is emphasized by those tumors which belong to the melanotic group but which contain no melanin. Perhaps the tumors here considered are also "amelanotic melanomas." This would lead one to hypothecate their origin from the chromatophore cells. At any rate it is obvious that they are quite different from the rapidly growing melanomas which spring from congenital pigmented moles. Since the whole problem of the nature and genesis of the melanotic tumors is still so imperfectly understood speculation along this line is not profitable. It is interesting to note, however, that certain new factors may need to be taken into account in our study of sarcomas. In 1918 (Hemorrhagic Degeneration of Myomas and Their Relation to Sarcoma, *J. Am. M. Asso.*, 1918, vol. lxxi, p. 1040), I called attention to the possibility of the development of sarcoma from the large mononuclear cells

FIG. 11.—Wen on the outer surface of the thigh. The surface of the tumor is covered with an attenuated skin simulating the bald-headed tumors.

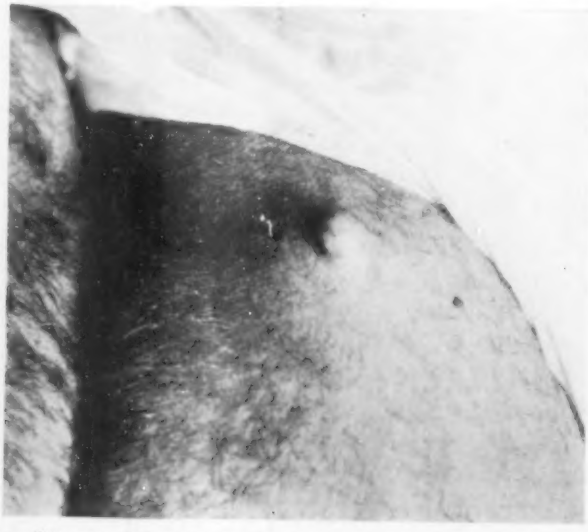


FIG. 12.—A flat lipoma, the size of a split orange, situated over the buttock. The teat-like projection has a reddened surface. This projecting part was made up of spindle cells while the larger part of the tumor was a typical lipoma.

from the blood stream. Just recently Carrel (Essential Characteristics of a Malignant Cell, *J. Am. M. Asso.*, 1925, vol. lxxxiv, p. 157) has shown that the large mononuclears and the macrophages represent the active elements in certain types of sarcoma. It is quite possible that we will soon be able to separate the sarcomas into those derived from a regression of adult tissue and those derived from cells native to normal blood. When that time comes the tumors now under consideration will be found to belong to the former group.

At present we can but say that the tumors considered in this paper differ from the conventional sarcomas by their slow growth, their point of origin and their late metastasis and that when they do metastasize they elect the lymphatic channels. This emphasizes anew the fact that it is not what a tumor is called, but what it does that is important to the surgeon. Whatever may be the nature and source of origin of these tumors the important fact to the surgeon, when he stands tumor in hand, is that he recognizes its nature and that a careful wide excision shall be made.

CONCLUSIONS

1. Certain tumors about the trunk are characterized by intimate attachment to the skin which gives them a peculiar and characteristic appearance.

2. They are of slow growth, spindle-celled fibrosarcomas in structure and metastasize by way of the lymphatics.

3. A properly performed operation results in a permanent cure.

4. That their relation to the general group of sarcomas suggests that they are derived from adult tissue.

EXOPHTHALMIC GOITRE AND TOXIC ADENOMA

SIMILARITY OF RESPONSE TO IODINE

By Allen Graham, M.D.

AND

Elliott C. Cutler, M.D.

OF CLEVELAND, OHIO

FROM THE DEPARTMENT OF SURGERY, THE LAKESIDE HOSPITAL AND THE WESTERN RESERVE UNIVERSITY SCHOOL OF MEDICINE

Recent American surgical literature conveys the impression that exophthalmic goitre and toxic adenoma are two different diseases. The differentiation is said to depend upon certain distinctive clinical, pathological and therapeutic observations. We have been led to question seriously the validity of this conception because our own experience has not confirmed such differential findings.

We have discussed previously [1] the clinical manifestations and anatomical and histological alterations in the thyroid in exophthalmic goitre and "toxic adenoma." Any clinical and pathological differences we have been able to recognize have not appeared to be of such fundamental significance as to establish these two clinical states as distinct diseases. These aspects of the subject will not be considered further at this time.

The purpose of the present paper is to point out the similarity of the response to iodine, as a measure preliminary to operation, in a series of cases of exophthalmic goitre and "toxic adenoma," selected in accordance with certain definite requirements for comparison.

Although it is well established that iodine is distinctly beneficial as a pre-operative preparation for thyroidectomy in cases of exophthalmic goitre, its use in any manner whatever in cases of "toxic adenoma" has been specifically condemned.[2]

The following impressions, gained from current literature, seem to reflect present-day views concerning this subject, and are set forth here as matters of interest in connection with observations we have to present:

1. Iodine is being used as a pre-operative preparation in surgical clinics, generally in cases of exophthalmic goitre but not in cases of toxic adenoma.

2. Exophthalmic goitre seems to be regarded as an essential dysthyroidism in the sense of an intoxication resulting from excessive quantities of perverted or incompletely iodized thyroid secretion.

3. Toxic adenoma seems to be regarded as an essential hyperthyroidism in the sense of an intoxication resulting from excessive quantities of normal or completely iodized thyroid secretion.

4. There appears to be a dilemma in certain cases that are said to have both exophthalmic goitre (dysthyroidism) and toxic adenoma (hyperthyroidism) at the same time. Iodine is known to be beneficial as a pre-operative preparation in the former, but is said to be contra-indicated in the latter.

5. Of particular interest and possible significance are certain cases in which clinicians of experience have made a definite clinical diagnosis of toxic adenoma, have withheld iodine before operation in view of that diagnosis, and have been astonished to observe a "typical post-operative hyperthyroid reaction" and even death following thyroidectomy. The unexpected post-operative reaction, or death, seems to have been accepted as a clear indication of mistaken pre-operative clinical diagnosis which was then changed accordingly from toxic adenoma to exophthalmic goitre. The change of diagnosis seems to have been supported further by the finding of certain histological alterations in the thyroid supposed to be characteristic for exophthalmic goitre (a supposition we regard as not well founded). The failure to utilize iodine as a preparation for thyroidectomy has been a source of chagrin in such cases.

6. The post-operative mortality is higher in cases of toxic adenoma than in cases of exophthalmic goitre.

7. There appears to be a third disease, "iodine hyperthyroidism," [3] which is neither exophthalmic goitre nor toxic adenoma. (With this disease the writers have had no personal experience. At all events it has no place in the present discussion since it is neither exophthalmic goitre nor toxic adenoma.)

Material.—In a series of fifty consecutive admissions to the general surgical division of The Lakeside Hospital, there are thirty cases of toxic goitre and twenty cases of non-toxic goitre. Among the thirty cases of toxic goitre there are twenty of the exophthalmic type and ten which correspond to what is called toxic adenoma.

For the purpose of comparing the response to iodine as a pre-operative preparation for thyroidectomy in cases of exophthalmic goitre and toxic adenoma, we have selected, from the above series, all cases that conformed to the following standard requirements:

1. Primary admissions to the general surgical service without previous operative intervention, iodine treatment, X-ray or other definite therapeutic regimen.

2. Two or more pre-operative and one or more post-operative determinations of basal metabolic rate.

3. The administration of iodine (Lugol's solution) before operation.

4. Primary thyroidectomy following the administration of iodine.

5. Uncomplicated by active infectious disease, notably tuberculosis.

In accordance with the above requirements we have for comparison ten cases of exophthalmic goitre and four cases of toxic adenoma (Charts 3, 4, 5, 6). It is hoped that this rigorous selection permits us to deal more accurately with the question at hand, than is often the case. Cases not selected for comparison of the two groups as specified above, may be utilized for the purpose of further illustrating the reaction to iodine under variable conditions of dosage, time relations, influence of previous therapy, state of the thyroid, and hospitalization (Charts 1, 2, Table I).

TABLE I

	Chart I		Chart II
	Case A	Case B	Case C
First admission (Medical)			
Days in hospital	31	16	25
Basal metabolic rate			
Admission	+50%	+44%	+59%
Discharge	+29%	+ 6%	+ 1%
Iodine (Lugol's solution)			
Hospital day started	15	1	8
Number of days given	17	16	18
Minims per day	15	15	30
Total quantity (minims)	255	240	540
Interval between admissions			
Days	6	21	32
Iodine	none	none	none
(*Second admission (Medical)*			
Basal metabolic rate			
Admission	+70%	+48%	+28%
On transfer to surgery	+29%	+24%	+46%
Pre-operative	+29%	+24%	+40%
Post-operative	+ 3%	−3%	+40%*
Iodine (Lugol's solution) pre-operative			
Hospital day started (Medical Service)	5	4	2
Number of days given (Medical Service)	6	5	31
Minims per day	45	10	20 to 30
Total quantity (minims) (Medical Service)	270	50	800†
Total quantity (Surgical Service)	225	30	none‡
Post-operative reaction	slight	slight	marked
Hospitalization (days)			
Iodine started (Medical Service)	5	4	2
Transfer to Surgical Service	10	9	46
Hospital day of operation	14	12	51
Surgical day of operation	5	4	6
Total hospital days (Medical and Surgical)	24	24	68

*See legend for explanation.
†Discontinued on the Medical Service fourteen days before transfer.
‡Not resumed on the Surgical Service.

TABLE I.—Details of three cases (A, B, and C) of exophthalmic goitre, the basal metabolic rate curves of which are shown in Charts 1 and 2, that have had two or more previous admissions to the medical service before transfer to surgery. Lugol's solution was administered during the medical hospitalization but not during the intervals between admissions.

The following points of interest are illustrated:

1. The failure of iodine to cure exophthalmic goitre even though the basal metabolic rate decrease to within normal limits after variable quantities of the drug for variable lengths of time.

2. The more favorable response to iodine when given for the first time as compared with the response to its use subsequently. (Compare Chart 3 with Charts 1 and 2).

3. Of two cases receiving the same quantity of iodine per day, and for about the same number of days, a greater percentage reduction of basal metabolic rate occurred in the case (Chart 1, curve B) in which iodine was instituted on the first hospital day, as compared with the case (Chart 1, curve A), in which iodine was instituted on the fifteenth hospital day.

4. On receiving iodine for the second time, after intervals without iodine varying from six to thirty-two days, the three cases show a decrease of basal metabolic rate accompanied by a corresponding improvement of clinical manifestations. In two cases (Chart 1) iodine was given nine and eight days, respectively, on the medical service and was continued on the surgical service to the date of operation, five and four days, respectively, after transfer. The response was as favorable as we usually see in previously untreated cases of equal severity. The post-operative recovery was equally favorable, although the technical difficulties of the operation appear to be increased in such cases as these.

Contrast the foregoing two cases with the third case (Chart 2) in which iodine was continued thirty-one days (second admission), a total of 800 minims of Lugol's solution being administered. Note the period of primary improvement followed by well-defined secondary rise due to the prolonged use of iodine in large quantities. Iodine was discontinued on the medical service, fourteen days before transfer and was not resumed on the surgical service. This we believe to be a mistake in surgical judgment. There was a severe post-operative reaction. Wound infection and the development of a foul sacral abscess complicated the post-operative course. On discharge, the basal metabolic rate was at the same level as before operation. Three months after discharge from the hospital the basal metabolic rate was normal and the patient had gained forty pounds in weight.

Concerning the differentiation of the two special groups compared, the following may be stated:

1. *Exophthalmic Goitre.*—Exophthalmos was present in most cases. There was diffuse parenchymatous hypertrophy and hyperplasia of the thyroid. The thyroid was not adenomatous (clinically), although small adeno-

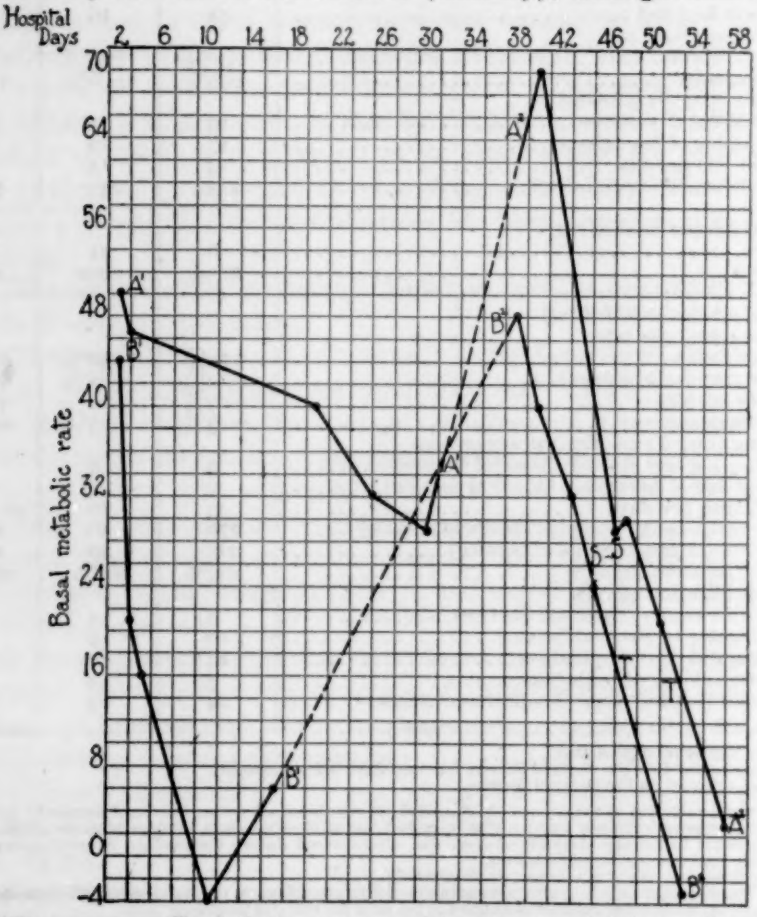


CHART 1.—————Hospitalization. — — — —Interval between admissions. S Transfer from medical service. T Thyroidectomy. Basal metabolic rate curves (A^1A^1 and B^1B^2) of two cases of exophthalmic goitre. Observation period is about two months, during which time there were two admissions to the medical service with final transfer to surgery and primary subtotal thyroidectomy. Case A had no previous treatment. Case B had a previous medical admission of thirty-nine days, during which time, with rest and luminal without iodine, the basal metabolic rate diminished from 41 per cent. above normal to 3 per cent. below normal. During the succeeding eight months of observation in the out-patient department a series of eight X-ray treatments, but no iodine was given. Further details of these two cases are shown in the first two columns of Table I.

mata were recognized, after removal, in a few glands. Thrill and bruit were present. Nervous and vasomotor phenomena were prominent features. Tachycardia, palpitation, increased appetite, loss of weight and strength were well-marked symptoms.

2. *Toxic Adenoma.*—Exophthalmos was absent in all cases. Adenomatous goitre was the outstanding clinical feature. Nothing comparable to the diffuse parenchymatous hypertrophy and hyperplasia that are said to

characterize exophthalmic goitre were present. There was no thrill or bruit. Nervousness, vasomotor disturbances, increased appetite, loss of weight and strength, while present in mild degree and in various combinations, in some cases, were not striking clinical features.

Exophthalmic Goitre.—The administration of iodine as a measure preliminary to thyroidectomy in cases of exophthalmic goitre is now a well-established procedure. Our experience is in accord with this principle. The

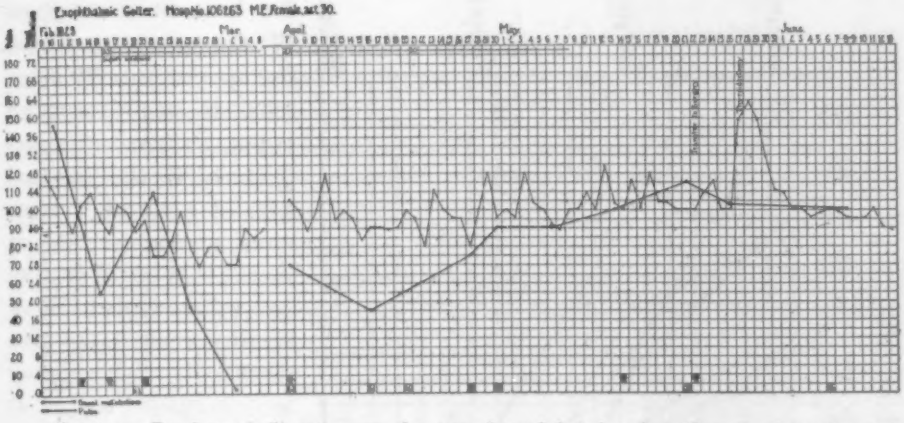


CHART 2.—Basal metabolic rate curve of a case of exophthalmic goitre (Case C) observed over a period of about four months, during which time there were two admissions to the medical service with final transfer to surgery and primary subtotal thyroidectomy. The interval between the two medical admissions was thirty-two days. The medical treatment consisted of rest, luminal and Lugol's solution during both admissions. Note the difference in response when iodine was administered for the first time as compared with its use subsequently. During the second admission there was a slight primary improvement, followed by a well-defined secondary aggravation, due to the prolonged use of iodine. Further details of this case are shown in the third column of Table I.

following factors appear to be of importance in determining the degree of clinical response:

1. The age and physical state of the patient.
2. The intensity and duration of the disease.
3. The physical and chemical state of the thyroid gland at the beginning of iodine therapy.
4. The degree of elevation of basal metabolic rate above normal.
5. Whether or not the patients previously have been taking iodine.
6. The presence of complicating conditions—tuberculosis, pyogenic infection, pregnancy, diabetes, nephritis, cardiac disease, etc.

The quantity of iodine necessary to accomplish the maximum clinical benefit previous to operation depends upon the combination of factors above outlined. Patients that have had iodine before coming to the surgeon show great variations in the degree of response to pre-operative iodine, and equally great variations in the quantity of iodine necessary to bring about a condition in which operation is safe. Under such circumstances the surgeon may be deprived of practically all the immediate advantages of pre-operative iodine and is confronted with the alternatives of performing a hazardous thyroidectomy, or resorting to procedures of lesser magnitude such as ligations or hemithyroidectomy, or of waiting for a period before re-instituting complete iodine therapy. Fortunately such cases constitute only a small percentage

of the total and are practically limited to those that have been taking iodine in large quantities or for a prolonged period of time immediately before admission to the surgical clinic. In such cases iodine should not be discontinued entirely (Chart 2), but thyroidectomy should be performed as early as possible or a lesser procedure resorted to in accordance with the clinical judgment of the surgeon.

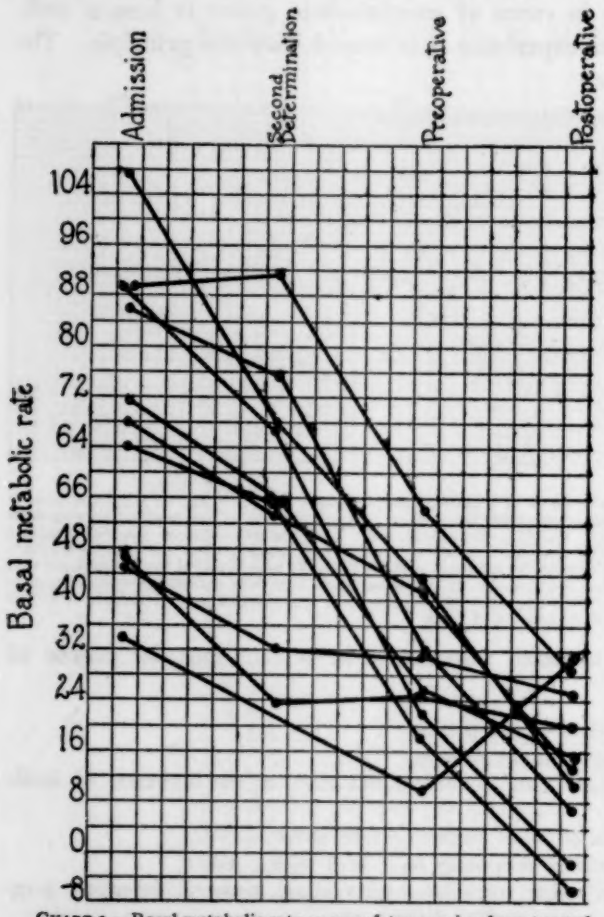


CHART 3.—Basal metabolic rate curves of ten previously untreated cases of exophthalmic goitre, showing typical response to iodine as a pre-operative preparation for thyroidectomy. The basal metabolic rates charted are: The first and second determinations after admission; the last determination before operation; and the average basal metabolic rate after operation, while still in the hospital. Further details are shown in Chart 5 and Table II.

Cases in which there has been an interruption of the previous iodine therapy are usually amenable to pre-operative iodine (Chart 1), particularly if the interval without iodine has been three to four weeks or longer. When this free interval amounts to three months or more, the previous iodine therapy may be disregarded so far as the resumption of iodine as a pre-operative measure is concerned.

In those hospitals where patients with exophthalmic goitre are admitted to the medical service and are to be transferred to surgery for operation, iodine therapy may be instituted on the medical service, but the surgeon should have the opportunity of selecting the time for operation and the type of operative procedure. With a proper coöperation on the part of the two services neither the patient, the internist, nor the surgeon need necessarily be at any particular disadvantage, and in some respects the patient's ends may be better served.

There is little doubt that from the standpoint of the surgeon the most acceptable cases of exophthalmic goitre are those that come under observation without previous treatment of any kind (Chart 3). In such cases a carefully planned pre-operative preparation, including the administration of iodine, permits a primary thyroidectomy in a very high percentage of cases and is

followed by a minimum of post-operative reactions. Although the hospitalization is somewhat increased by reason of the iodine therapy, repeated admissions are minimized, incomplete and multiple stage operations are largely done away with, the maximum surgical therapy is accomplished in a shorter period of time, and the patients have the advantage of earlier restoration of their economic status.

Toxic Adenoma.—Within the past few years "toxic adenoma" as an entity, in contra-distinction to exophthalmic goitre, seems to have met with increasing acceptance. The basis for differentiation depends in no small measure upon a supposed directly opposite response to iodine in the two instances. Indeed the administration of iodine has been proposed as a method of making the distinction in borderline or doubtful cases.[4] Furthermore, the condition called toxic adenoma is said to be brought about by the administration of iodine to borderline toxic cases or to cases of simple endemic goitre with adenomatous thyroids. The above tenets are in many respects so contrary to our experience that it seems worth while to discuss the matter in some detail.

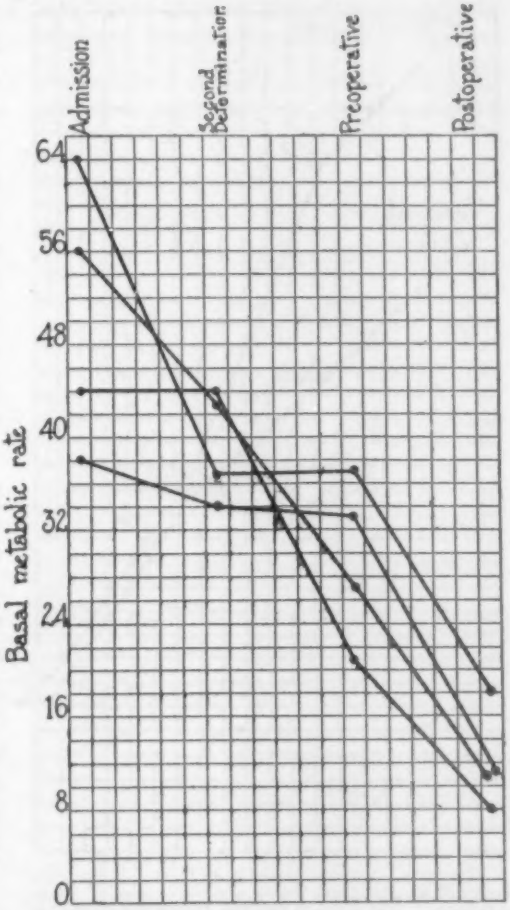


CHART 4.—Basal metabolic rate curves of four previously untreated cases of "toxic adenoma," showing the response to iodine as a pre-operative preparation for thyroidectomy. The basal metabolic rates charted are: The first and second determinations after admission; the last determination before operation; and the average basal metabolic rate after operation, while still in the hospital. Further details are shown in Chart 5 and Table II.

We know of no symptoms, physical signs, nor alterations in the thyroid which singly are necessarily pathognomonic for the condition called "toxic adenoma," *i.e.*, that are invariably present in toxic adenoma and invariably absent in exophthalmic goitre or simple endemic goitre.[1]

We recognize fully that patients with adenomatous thyroids and increased basal metabolic rates, come to the clinic with the history of having taken iodine as medicine or as iodized table salt, and with the history that their condition has been made worse thereby. From such cases one might readily conclude that iodine caused the disease. Careful inquiry usually discloses the

presence of definite symptoms preceding the institution of the iodine. However suggestive these cases may be, it is probably not without significance that a history of previous iodine therapy can be obtained in less than half of those cases of "toxic adenoma" coming under observation. Moreover, it is to be noted that the same circumstances and sequence of events are frequently encountered in cases of exophthalmic goitre with and without adenomatous thyroids. Our deliberate efforts to produce the clinical state called toxic adenoma by the administration of large quantities of iodine to borderline toxic cases and cases of simple endemic goitre with adenomatous thyroids have been uniformly unsuccessful.

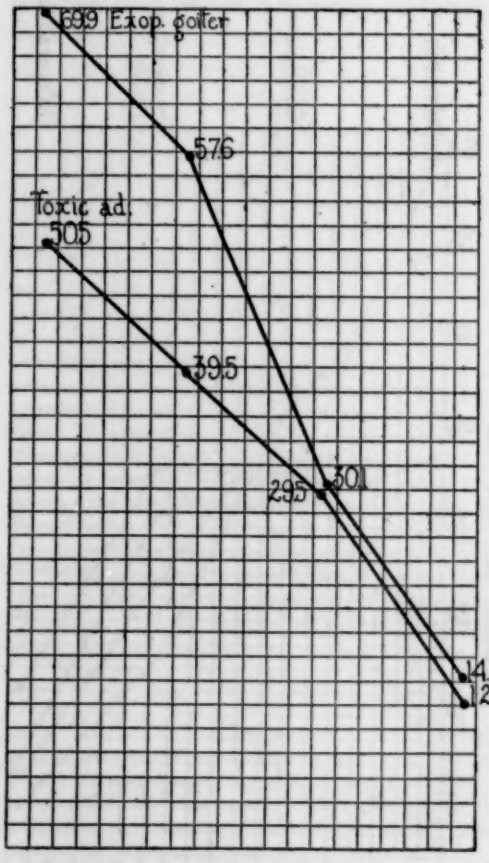


CHART 5.—Average basal metabolic rate curves of ten cases of exophthalmic goitre and four cases of "toxic adenoma." The cases in the two groups are selected in accordance with certain definite requirements for comparison (*vide* text). The metabolic rates charted are: The first and second determinations after admission; the last determination before operation; and the average basal metabolic rate after operation, while still in the hospital. The parallelism of the two curves is striking. Further details are shown in Table II.

Much emphasis has been placed upon the observation that in cases of established toxic adenoma the clinical condition is aggravated by the administration of iodine. A careful consideration of the reaction to iodine that takes place in cases of exophthalmic goitre, we believe, offers an adequate explanation of the phenomena observed in cases of toxic adenoma under similar circumstances.

As previously noted, cases of exophthalmic goitre that receive iodine for the first time are definitely improved up to a certain point, as indicated by a decrease of basal metabolic rate (Chart 3) and amelioration of the clinical symptoms. If iodine be continued in the same dosage for a sufficiently long period of time, the clinical condition becomes aggravated and the basal metabolic rate (Chart 2) may increase to its original level or even to a higher level.

In cases of toxic adenoma that come under observation after taking iodine, with a history that the clinical symptoms have been made worse, the question naturally suggests itself: are we not seeing these cases in the period of secondary aggravation that follows a period of primary improvement, similar

to that occurring in cases of exophthalmic goitre. In *previously untreated* cases of toxic adenoma, according to our experience, such a period of primary improvement (Chart 4) following the administration of iodine has been the rule and not the exception.

Concerning Dysthyroidism in Exophthalmic Goitre and Hyperthyroidism in Toxic Adenoma.—We have observed that classical cases of exophthalmic goitre, receiving iodine for the first time, show a period of primary improvement (Charts 2, 3, 6) followed by a period of secondary aggravation (Charts

TABLE II

	Exoph. goitre average 10 cases	Toxic adenoma average 4 cases
Basal metabolic rate		
Admission	+69.9% (2.4)*	+50.5% (2)
Second	+57.6% (5)	+39.5% (3.25)
Pre-operative	+30.1% (18.2)	+29.5% (8.5)
Post-operative	+14.2% (30.7)	+12.0% (18)
Iodine (Lugol's solution)		
Number days given	16.2	7.5
Minims per day	28.0	22.6
Total quantity, pre-operative	452.2	69.5
Hospitalization		
Day iodine therapy started	4.6	3.5
Day of operation	20.8	11.0
Total hospital days	33.3	19.25

*Numbers in parenthesis represent hospital days.

TABLE II—Details of Charts 3, 4 and 5, showing the relations of time (hospital days), daily dosage and quantity of iodine (Lugol's solution) to basal metabolic rate in previously untreated cases of exophthalmic goitre and "toxic adenoma" receiving iodine as a pre-operative preparation for primary thyroidectomy.

2, 6), provided the iodine is continued in the same dosage and for a sufficiently long period of time.

We have observed that *previously untreated* cases of toxic adenoma improve in about the same ratio as cases of exophthalmic goitre (Chart 5, Table II), following the administration of iodine. They also show a period of secondary increase of basal metabolic rate (Chart 6) when iodine is continued. We have not actually carried out the experiment of continuous iodine therapy in cases of toxic adenoma to the point of obtaining such a degree of secondary aggravation as has been observed by us in cases of exophthalmic goitre following the prolonged use of iodine.

If exophthalmic goitre be a pure form of dysthyroidism—in the sense of an intoxication resulting from an over-production of a perverted or incompletely iodized thyroid secretion—and is greatly improved by the administration of iodine, what explanation do we have for the secondary period of aggravation when iodine is continued? It might be urged that the disease exophthalmic goitre (without adenomatous thyroid) can be converted from a state of dysthyroidism into a state of hyperthyroidism by the use of iodine. If this be so, it would seem logical to expect relief by continuing the drug. Experience has shown that the sudden stopping of iodine

(Chart 2) in cases of exophthalmic goitre that have taken the drug for a long time or in large quantities is rarely followed by any striking clinical improvement and frequently the clinical condition becomes worse. We wish

TABLE III

	Exoph. goitre average 4 cases		Toxic adenoma average 2 cases	
Hospitalization:				
Day of operation	30.0		12.5	
Total hospital days	46.25		18.5	
Iodine (Lugol's solution)				
Hospital day started	5.5		3.5	
Minims per day, period of improvement	25.54		21.0	
Total quantity, period of improvement	319.0	(12.5)*	52.5	(2.5)
Total quantity, period of secondary rise	247.5	(7.5)	82.5	(4)
Total quantity, pre-operative	713.5	(24.5)	196.5	(9)
Basal metabolic rate				
Admission	+76.5%	(2.75)	+50.0%	(2)
At start of iodine	+65.0%	(3.5)	+43.5%	(3)
Lowest, period of improvement	+24.75%	(18)	+18.5%	(6)
Highest, period of secondary rise	+39.5%	(25.5)	+24.0%	(10)
Post-operative	+15.7%	(42)	+ 9.5%	(18)
Reduction of basal metabolic rates, period of improvement				
Per cent	61.9		57.5	
Days required	14.5		3.0	
Lugol's required, minims	319.0		52.5	
Increase of basal metabolic rate, period of secondary rise:				
Per cent	55.5		30.0	
Days required	7.5		4.0	
Lugol's required, minims	247.5		82.5	
Ratio of basal metabolic rates				
On admission	100.0		65.4	
At start of iodine therapy	100.0		66.9	
Lowest, period of improvement	100.0		74.7	
Highest, period of secondary rise	100.0		60.7	
Ratio of maximum reduction of basal metabolic rate during period of primary improvement				
Lugol's required	6.0		1.0	
Days required	5.0		1.0	

*Numbers in parentheses represent hospital days.

TABLE III.—Details of four cases of exophthalmic goitre and two cases of "toxic adenoma" the average basal metabolic rate curves of which are shown in Chart 6. In these cases a period of primary improvement and a period of secondary rise following the administration of iodine are illustrated. Although the response to iodine is of the same character in cases of exophthalmic goitre and "toxic adenoma" there is a striking contrast in regard to the quantity of iodine required and the number of days necessary to accomplish the same result in the two groups.

to emphasize that the supposed dysthyroidism, hyperthyroidism and the conversion from one state to the other, in the hypothetical case above, occurs in a patient who has not an adenomatous thyroid.

If toxic adenoma be a pure form of hyperthyroidism, in the sense of an intoxication resulting from an over-production of normal or completely iodized secretion, what explanation have we for the period of primary

improvement (Chart 4) noted in *previously untreated cases* in which iodine is administered? It might be urged that during this period of improvement the patient is in a state of dysthyroidism. How can we reconcile the dysthyroidism with the conception that toxic adenoma is a pure form of hyperthyroidism?

Concerning the question of dysthyroidism or hyperthyroidism, and the relation of these states to iodine therapy, the material considered in this paper

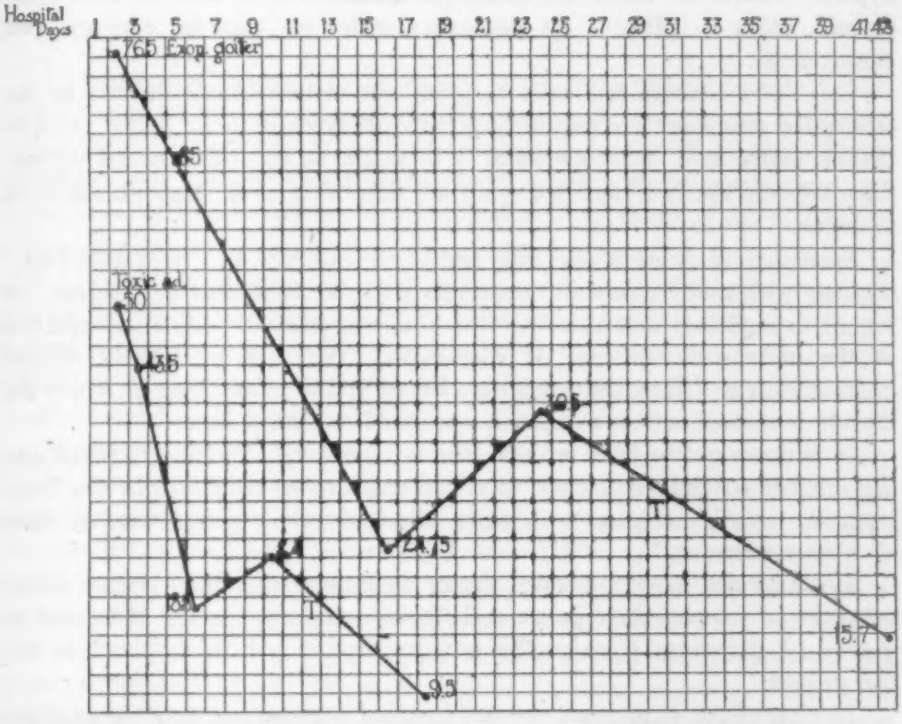


CHART 6.—Average basal metabolic rate curves of four cases of exophthalmic goitre and two cases of "toxic adenoma" in which a period of secondary rise succeeds the period of primary improvement following the administration of iodine. All of the cases are taken from the groups represented in Charts 3 and 4. The basal metabolic rates charted are the following: 1. On admission. 2. At the beginning of iodine therapy. 3. Lowest rate during the period of primary improvement. 4. Highest rate during the period of secondary rise. 5. The average basal metabolic rate following operation. The two curves are practically identical in character. Time (hospital days) and the quantity of iodine are the variable factors of significance. See Table III.

affords no satisfactory basis for distinction between exophthalmic goitre and toxic adenoma.

Our cases of toxic adenoma were not only not aggravated by the administration of iodine, but responded in the same manner and in about the same ratio as did the cases of exophthalmic goitre. *We emphasize again that both groups were previously untreated.*

COMMENT

We appreciate the inadvisability of making generalizations from a small number of observations. However, our experience in this field is more extensive than is represented by the small series of cases here considered. It

is proper to state that in the more extended experience we have found nothing fundamentally inconsistent with the observations that form the basis of this paper. In fact, the material considered constitutes an objective confirmation of the views we have previously held.

In this paper we have not been particularly concerned with the question of whether toxic goitre, in its broadest sense, is an essential dysthyroidism, hyperthyroidism or both; whether toxic goitre is a disease in which the thyroid plays a primary or secondary rôle, as regards etiology and pathogenesis.

Our chief concern has been to point out certain inconsistencies in the conception that there are two directly opposed types of toxic goitre (exophthalmic goitre and "toxic adenoma") in so far as any fundamental distinctions between the two supposed types are dependent upon opposite reactions to iodine.

Increased basal metabolic rate and other evidence of "thyrotoxicosis," together with hypertrophy and hyperplasia of the thyroid of any degree, we regard as sufficient indication for the administration of iodine, irrespective of the presence or absence of adenomata. We shall not expect clinical improvement to follow the administration of iodine in any case in which the thyroid is normal or in a completely involuted or colloid state.

It is important to bear in mind that the quantity of iodine required and the number of days necessary to effect comparable reduction in the basal metabolic rate is far greater in cases of exophthalmic goitre than in cases of "toxic adenoma."

Probably the most important factor in determining the relative iodine tolerance of exophthalmic goitre and "toxic adenoma" is the difference in degree of glandular hypertrophy and hyperplasia of the thyroid in the two groups.

In view of our experience, we feel justified in recommending the administration of iodine as a measure preliminary to operation in all cases of toxic goitre whether the thyroid be adenomatous or non-adenomatous.

REFERENCES

[1] Graham, A.: The Thyroid Gland in Relation to "Toxic Goitre," Radiology, May, 1926, vol. vi, p. 377; A Consideration of Certain Phases of the Problem of Graves' Disease, Read at the Annual Meeting of the American Association for the Study of Goitre, Louisville, Kentucky, February 1, 1926, Accepted for Publication in Chicago Medical Recorder; Exophthalmic Goitre and Toxic Adenoma. Clinical Variations of a Single Disease, J. A. M. A., in Press; Cutler, E. C., and Graham, A.: Toxic Goitre, S. Clinics, N. America, in Press.

[2] Boothby, W. M.: The Use of Iodine in Exophthalmic Goitre, Endocrinology, November, 1924, vol. viii, p. 727.

[3] Jackson, A. S.: Iodine Hyperthyroidism, Boston M. and S. J., December 17, 1925, vol. cxciii, p. 1138.

[4] Kimball, O. P.: Functional Test for Differential Diagnosis of Hyperthyroidism, S. Clinics, N. America, August, 1924, vol. iv, p. 981; Differential Diagnosis of Hyperthyroidism, Ohio State M. J., July, 1924, vol. xx, p. 421.

THE FREQUENCY AND CHARACTER OF BLADDER DISTURBANCES IN NEWGROWTHS OF THE BRAIN AND SPINAL CORD*

By Charles A. Elsberg, M.D.
of New York, N. Y.

In organic diseases of the brain and spinal cord, there are many factors that may and do have an influence upon the proper functioning of the bladder and urethra—disturbances in the psychic sphere in lesions of the frontal lobes and the corpus callosum; somnolence, stupor or coma that characterizes many intracranial affections and that regularly ensue in the terminal stages of intracranial disease; disturbances which are associated with polydipsia and polyuria, that follow especially in lesions of the hindermost parts of the pituitary body and of the floor of the third ventricle; interference with afferent visceral and somatic pathways for sensibility, and efferent pathways for motor control, which occur in affections of the spinal cord where more or less of the transverse diameter of the cord is involved.

The functions of the bladder are under the combined control of three mechanisms of which one is derived from the sympathetic system. The sympathetic connector fibres leave the spinal cord by the two lower thoracic and the two upper lumbar nerves (Holmes and Walshe) or perhaps the four upper lumbar nerves, and pass, with relays, to the hypogastric nerves and the bladder wall. The second nervous connection is not derived from the sympathetic system, but directly from the second, third and fourth sacral nerves, and passes to the wall of the bladder by way of the nervi erigentes. The third, and perhaps voluntary element in the nervous mechanism of micturition passes by the pudic nerves to the urethral muscles.

Voluntary micturition and evacuation of the bladder is in some way controlled by the brain through the nervi erigentes and the pudic nerves, and results from simultaneous contraction of the bladder musculature and a relaxation of the sphincter muscles of the neck of the bladder and urethra.

Difficulty in the expulsion of urine may be due, aside from mechanical obstruction, to a diminution in the muscular power of the bladder, an increase in the tone of the sphincter muscles, or a combination of these two factors, from a disturbance in non-sympathetic nervous control, while true vesical incontinence in the conscious individual is probably rare excepting when all three mechanisms are interfered with.

The control of bladder function may, however, be exercised by the spinal cord when part of the cord has been entirely separated from higher centres, although such bladder activity is purely reflex and not under voluntary control.

* Read before the New York Academy of Medicine, February 18, 1926.

In the following table (Table I) is given a general classification of the nature of a possible disturbance in the nervous control of the bladder and urethra, and its effect upon bladder function:

TABLE I

Nature of Disturbance of Nervous Control of Bladder and Urethra (1–4 Cerebral; 5–13 Spinal)	Effect upon Bladder Function
1. Diminished psychic control.	1. Conscious voluntary micturition, but not controlled as to time and place.
2. Loss of psychic control.	2. Unconscious (?) voluntary micturition not controlled as to time and place.
3. Diminished voluntary control (consciousness diminished).	3. Unconscious (?) reflex involuntary micturition.
4. Loss of voluntary control (consciousness lost).	4. Unconscious reflex involuntary micturition.
5. Partial paralysis of bladder musculature.	5. Dysuria, incomplete emptying of bladder, perhaps increased frequency of urination.
6. Total paralysis of bladder musculature.	6. Retention, perhaps overflow (false incontinence).
7. Paralysis of musculature of urethra and neck of bladder.	7. Frequent conscious involuntary micturition.
8. Paralysis of musculature of urethra and of bladder.	8. Involuntary dribbling of urine.
9. Increased irritability of bladder musculature.	9. Increased frequency of micturition.
10. Diminished irritability of bladder musculature.	10. Partial or complete retention.
11. Diminished visceral sensibility of bladder (diminished transmission of afferent impulses).	11. Same as 10. Sometimes increased frequency.
12. Loss of visceral sensibility of bladder.	12. Retention and overflow.
13. Diminution or loss of somatic sensibility of urethra.	13. Unconscious passage of urine.

1. *Disturbances of Micturition in Tumors of the Brain.*—Increased frequency of urination, difficulty in urination, retention or incontinence may occur from intracranial expanding lesions, but are much less frequent than in newgrowths of the spinal cord. If the patients who are in stupor or coma are excluded, bladder disturbances occur in only one-fourth of the patients, and in not a few of them, the urinary difficulty is, at least in part, the result of a clouding of consciousness or a psychic defect. In some of the patients, however, increased frequency of micturition is due to an increased renal activity; the patient voids more often because of the larger amount of urine that is secreted, and such an individual with a polyuria, may have periods of nocturnal incontinence.

A story of attacks of loss of bladder control is often obtained from patients with intracranial newgrowths, but this incontinence is usually an overflow

from retention rather than a true incontinence. The figures, from our clinic, of the relative frequency of vesical incontinence ought to be and would be much smaller, if it were possible to distinguish in the patient's history, between a false incontinence due to overflow, and *sensu strictu* the loss of control due to a psychic disturbance or to an actual weakness or paralysis of the vesical and urethral musculature.

Doctor Gould has collected for me the urinary disturbances recorded in 165 patients with verified tumors of the brain. Sixty patients (36 per cent.) had some disturbance of normal micturition, and 39, or 23 per cent., had occasional or continued incontinence. Sixty-nine of the 165 patients (41 per cent.) had a more or less marked mental disturbance (Table II).

TABLE II

History of Mental and Urinary Disturbances in 165 Patients with Brain Tumors

Location	Number	Mental disturbances	Urinary disturbances	Incontinence occasional or persistent	Dysuria	Increased frequency
Frontal	40	36	29	19	4	4
Motor	3	1	1	1	0	0
Parietal	22	8	5	3	2	4
Temporal	19	11	5	4	2	4
Occipital	4	2	3	2	1	1
Unlocalized	3	0	0	1	1	1
Pituitary and interpeduncular	10	3	2	1	0	2
Intraventricular	4	0	2	2	0	0
Cerebellar and cerebello-pontine angle	60	8	13	6	8	3
Totals	165	69 41%	60 42%	39 23%	18 11%	19 12%

In tumors of the frontal lobes mental disturbances were, of course, very frequent (36 of 40 patients). Most of the patients had some variety of disturbance of bladder function, incontinence of urine being very frequent. Thus three-fourths of the patients with growths in the frontal lobes had urinary disturbances of one kind or another, while almost 50 per cent. had occasional or continued incontinence. In comparison, incontinence was comparatively rare when the growth was in some other part of the brain; it occurred in only 21 per cent. of the patients with disease in the temporal lobe, 14 per cent. of those with disease in the parietal lobe and 10 per cent. of those with growths in the posterior cranial fossa. When the tumor was in the left cerebral hemisphere, incontinence was more frequent than when it was on the right. In other words, difficulty in and increased frequency of micturition occur fairly often in tumors of the brain, but, if patients in the terminal

stages of their disease be excepted, incontinence of urine is frequent only in frontal lobe growths.

If patients in stupor or coma or with marked mental changes were excluded, urinary disturbances were no more frequent in subcortical than in cortical growths (Table III).

TABLE III

Mental and Urinary Disturbances Observed in Patients While in Hospital with Cortical as Compared to Those with Subcortical Growths (Patients in Coma Excluded)

	Cortical growths						Subcortical growths					
Location	Number	Mental disturbances	Urinary symptoms	Occasional incontinence	Dysuria	Increased frequency	Number	Mental disturbances	Urinary symptoms	Occasional incontinence	Dysuria	Increased frequency
Frontal	10	5	5	4	1	3	23	16	9	7	2	1
Motor	1	0	0	0	0	0	2	1	1	1	0	0
Parietal	9	1	1	1	2	2	11	6	2	1	0	1
Temporal	0	0	0	0	0	0	17	9	5	4	1	2
Occipital	2	1	1	0	1	0	2	0	0	0	0	0
Cerebellar	3	0	0	0	0	0	33	4	5	3	3	0
Cerebello-pontine angle	17	4	3	3	2	2	0	0	0	0	0	0
Totals	42	11 26%	10 24%	8 19%	6	7	88	36 41%	22 25%	16 19%	6	4

It is well known that mental changes are more frequent in patients with subcortical infiltrating tumors; in the series we studied, they occurred in 41 per cent. of the subcortical as compared with 26 per cent. of the cortical growths. It was somewhat surprising, however, to find that if patients with mental changes and those in stupor or coma in the terminal stages of their disease were excluded, disturbances of the bladder function were as frequent in superficial growths—endotheliomas, cerebello-pontine angle tumors, etc.—as in deeply situated infiltrating growths.

2. *Bladder Disturbances in Tumors of the Spinal Cord.*—Some years ago Doctor Stookey studied the material of my clinic and published the results of his investigations of bladder and rectal disturbances in cord tumors. The conclusions at which he arrived, have been modified in some respects by my study of a larger series of cases. Bladder disturbances occur much more frequently in extramedullary, extradural and conus and cauda tumors. Eighty per cent. of the extramedullary growths, 84 per cent. of the extradural growths, and 60 per cent. of the intramedullary growths had bladder disturbances, but all in all, a tumor outside of the substance of the cord is more

apt to cause bladder disturbances than one within the substance of the cord. (Table IV.)

TABLE IV

Frequency of Bladder Disturbances in 105 Spinal Cord Tumors

Extramedullary	54, bladder disturbances in 43 = 80 per cent.
Conus and cauda equina	23, bladder disturbances in 18 = 78 per cent.
Extradural	13, bladder disturbances in 11 = 84 per cent.
Intramedullary	15, bladder disturbances in 9 = 60 per cent.

There is no particular segment of the spinal cord that is especially concerned with control of the vesical sphincter. The lower down in the spinal cord the compression by the tumor, the greater is the frequency of bladder disturbances (Table V).

TABLE V

Frequency of Bladder Disturbances at Different Levels of the Spinal Cord

Cervical 1 to 4	10	bladder disturbances in 22 = 71 per cent.
Cervical 5 to 8	21	
Thoracic 1 to 6	34	bladder disturbances in 25 = 74 per cent.
Thoracic 7 to 12	26	bladder disturbances in 20 = 81 per cent.
Lumbo-sacral and cauda	15	bladder disturbances in 13 = 87 per cent.

Excepting in extradural primary or secondary malignant disease in which, with the rapid advance of the disease, bladder disturbances occur early, bladder disturbances usually appear late in spinal cord tumors. In the ordinary extramedullary tumors usually at least six to twelve months elapse before vesical disturbance is noticed, but there may be very little bladder disturbance even after many years, if the tumor be a slow growing one and the compression of the spinal cord not much advanced. The spinal centres to the bladder lie in the sacral segments of the cord, and it is interesting and somewhat surprising that even in tumors in this location at least a year will elapse before distinct bladder disturbances are observed. During the second year, however, bladder disturbances are more frequent and 60 per cent. of the patients have a more or less marked interference with bladder function.

In intramedullary tumors, bladder disturbances are observed in one-half of the patients within twelve months from the beginning of their symptoms. In other words, in those patients with disease within the substance of the cord, interference with bladder function although more rare, appears earlier (Table VI).

TABLE VI

Period Between First Symptoms of Tumor and Appearance of Bladder Disturbance

	Extra-medullary	Conus and Cauda	Extradural Malignant	Extradural Non-malignant	Intra-medullary
Less than 6 months	8 = 21%	1 = 21%	4 =60%	—	—
6 — 12 months	10 = 26%	2 = 20%	1	—	3 = 50%
1 — 2 years	12 = 28%	6 = 60%	1	3 = 43%	2 = 30%
2 — 3 years	3 = 8%	—	1	3 = 43%	1
More than 3 years	5 = 13%	1 = 10%	0	1	0

If the time of appearance of bladder disturbances be looked at from the viewpoint of the part of the cord that is affected (Table VII), we have found that in patients with high cervical tumors somewhat less than one-half have bladder disturbances before two years. In the lower cervical region, about three-quarters have similar disturbances before two years have passed. In the thoracic region, about four-fifths, and in the lumbo-sacral region, and cauda

TABLE VII

Time of Appearance of Bladder Disturbances from Viewpoint of Affected Level

	C 1–4	C 5–8	Th 1–6	Th 7–12	Lumbo-sacral and cauda
Less than six months	1	2	2	2	2
Six to twelve months	–	4	9	6	2
One to two years	2	3	7	4	6
Two to three years	4	3	1	1	0
More than three years	–	1	2	3	1

equina, ten-elevenths of the patients have such disturbances. In other words, this shows again the increasing frequency of disturbances of the bladder function in lesions of the lower parts of the cord.

We have further investigated the different kinds of bladder disturbance and the time of its appearance. Much will depend upon the rapidity of growth of the tumor and the amount of compression of the cord. A slow growing tumor will cause much less interference with cord function than will a more rapidly growing one, and a soft tumor will likewise cause less damage, and therefore be less apt to cause bladder symptoms than a growth which is of firm consistency. With these reservations one may say the following. If the spinal symptoms have lasted less than six months, difficulty in emptying the bladder is most frequent; if they have lasted from six to twelve months, difficulty in urination or incontinence are frequent; and when the symptoms and signs of spinal compression have lasted several years or more, incontinence either due to overflow or true paralysis of the sphincter muscle, is most frequently observed (Table VIII).

That the degree of bladder disturbance depends not only upon the level involved, but also upon the degree of compression, is shown in Table IX. All types of bladder disturbances were rare whenever there was little motor or sensory disturbance, and the more marked the disturbances in power and sensation, the more frequent were the bladder disturbances and the more severe their nature. Thus, of 38 patients with complete retention or incontinence, 30 had marked or advanced motor and sensory disturbances. A few patients with distinctly marked involvement of power and sensation had normal bladder function, but the majority had very marked disturbance of vesical control as soon as sensory and motor signs were fairly advanced. In a few patients without much sensory disturbance but with distinct motor involve-

ment, there were bladder disturbances, but the rule seems to be that both sensory and motor functions must be considerably involved before marked bladder disturbances occur.

After the removal of the tumor the improvement in vesical control may occur very rapidly and a patient who has lost all control for many weeks

TABLE VIII

Type of Bladder Disturbance and Time of Appearance

	Dysuria	Frequent urination	Occasional incontinence	Complete incontinence	Retention
Less than six months	6	3	1	3	2
Six to twelve months	5	2	3	8	3
One to two years	7	3	2	10	2
Two to three years	1	1	2	2	
More than three years	1	1	4	5	

If symptoms lasted less than six months—dysuria most frequent
If symptoms lasted six to twelve months—dysuria or incontinence frequent
If symptoms lasted one to two years —incontinence most frequent
If symptoms lasted two to three years —incontinence most frequent
If symptoms lasted more than three years—incontinence most frequent

may within a few days recover considerable power and within a few weeks regain complete control of the bladder function. During the period that the patient is recovering his vesical control, the contraction of the bladder when distended with urine must be an imperfect one; not so rarely the patients are only able to partially empty the bladder and a number of ounces of residual

TABLE IX

Relation of Bladder Disturbances to Degree of Motor and Sensory Disturbance

	Dysuria, frequent urination	Incontinence or retention	No bladder symptoms
Slight motor and sensory	1	2	2
Distinct motor and sensory	19	4	9
Marked motor and sensory	18	18	8
Advanced motor and sensory	1	12	0
Motor much less than sensory	1	1	2
Sensory much less than motor	3	1	1
	43	38	22

urine remains. It would be very interesting for some one to study cystoscopically the contraction of the bladder which is improving after the removal of a cord tumor and while a few investigations on this subject have been made, they are far from convincing and much more careful studies are desirable.

It is very interesting also that visceral sensibility, the consciousness of bladder fulness reappears very quickly in the patients who improve, and it seems that this return of visceral sensibility precedes return of somatic sensation.

If the lesion of the spinal cord from the prolonged pressure of a tumor has been so great that no recovery of power is possible, then a pure spinal automatic activity may reappear after a few months, even when the sacral cord or roots of the cauda equina are involved. In general, however, in many of the conus or cauda lesions, true incontinence with relaxation of bladder and urethral musculature and continuous dribbling of urine persists. In the patients who had an irremediable cord lesion at a higher level, the threshold for contraction of the bladder is so much lowered that one can keep the patient comfortable and dry by causing automatic emptying of the bladder every few hours by external irritation such as pinching the thigh or sudden passive movement of a limb.

One must never forget that an individual with a brain or a spinal cord tumor may have a bladder disturbance which is in no way connected with the intracranial or spinal disease. I have seen patients with enlarged prostate and cystitis who had also a spinal cord tumor. There are individuals who either as a result of or not connected with bladder disturbances from their disease, develop calculi in the bladder or in the kidneys or lime incrustations in the bladder wall that result in marked urinary disturbances. Whenever, therefore, bladder disturbances persist after the relief of the intracranial or spinal lesion, the bladder should be carefully studied with the cystoscope for evidence of local disease.

ANALOGIES BETWEEN THE BILIARY TRACT AND THE URINARY TRACT*

By Edwin Beer, M.D.
of New York, N. Y.

A study of the similarities in the processes that take place in rather unrelated systems is liable to be of more than passing interest and may prove instructive and stimulating. Even though our knowledge be but surface gleanings, and though future investigations may upset much that we believe in, still certain striking analogies between pathological and clinical observations in the biliary and in the urinary tracts have struck me and have interested me for years, and to some of these I wish to refer in this very brief paper. I feel that a better appreciation of these analogies has given me a better understanding of the two systems, even though at times speculation rather than complete scientific proof supports the thesis.

There are certain rather self-evident similarities which must be mentioned to be dismissed. At the *fons et origo* of each system is a paired glandular organ—for the liver is much like the kidneys. It is in fundamental structure a fused or bi-lobed organ, each half, from an excretory standpoint at least, separate from the other. Both liver and kidneys have definite excretory functions and in both the excretion is poured into two ducts—to be stored in the two bladders and subsequently again passed through a duct into the outer world or into the intestine which communicates directly with the exterior.

Passing these anatomical and physiological similarities rapidly and turning our attention to the clinical and pathological, and surely more speculative problems, it must be admitted that despite the vast amount of investigation devoted to the biliary system, our knowledge of the urinary tract is far more complete. An intimate knowledge of the latter may throw some suggestive lights on processes that take place in the biliary tract that are less clear and are difficult to understand; and vice versa, an appreciation of what we have learned in the study of diseases of the liver and of the biliary tract, may assist us in interpreting our observations on the urinary tract.

In studying these similarities between the two systems, the subject naturally falls into several subdivisions and the analogies stand forth rather vividly —perhaps more vividly than they should. For purposes of this discussion, I have divided the available material under the following three heads: 1—Cholesteremia and uricemia; 2—lithiasis, (a) primary, (b) secondary; 3—infections; and will limit myself to a brief discussion of these as they illustrate rather cogently the analogies between the two tracts.

* Read by title before the American Surgical Association, May 26, 1926.

Cholesteremia and Uricemia.—Cholesterol may be considered as a more or less specific hepatic excretion and uric acid bears a somewhat similar relation to renal function even though both substances are widely distributed in the other body tissues. Both substances are regularly present in definite amounts in these respective excretions and the amount (or percentage) seems to vary with the concentration of these substances in the blood stream. For many years it was thought that the concentration in the blood stream could not be altered by diet and, consequently, diet was thought of questionable influence on the amounts of cholesterol or of uric acid excreted in the bile or in the urine. Gradually, experimental feeding experiments have shown that the blood concentration can be increased and with that the amounts excreted are also increased. On the one hand, the cholesterol content can be increased by eating a fatty diet, *e.g.*, egg-yolk, brains, cream, etc.; on the other, the uric acid content can be increased by eating foods rich in nucleins, *e.g.*, thymus, spleen, liver, lungs, kidneys, etc. Moreover, in certain diseases the blood content of these two substances is affected; for instance, the post-infective period of typhoid usually shows an increase in blood cholesterin, and the uric acid content is increased in pneumonia at the crisis. More interesting, however, is the observation of the influence of functional disturbances of the liver and of the kidneys on the blood concentration of these two substances. In bilateral renal obstruction, such as we see in early prostatic cases, the early evidence of functional impairment of the kidneys is found in an increase of uric acid in the blood. Similarly, in the biliary tract, one of the first signs of choledochus obstruction, incomplete or complete, is a heaping up of the cholesterol in the blood. In both cases with removal of the cause, the physiological disturbance is relieved and the blood chemistry returns to normal.

Whether in such and similar functional disturbances there are irregular fluctuations and that from minute to minute the concentration in the blood varies, is not known though it is probable. The occasional spilling over of concentrated solutions, of over-saturated colloidal solutions, might account for many of the obscure clinical pictures that have masqueraded here and abroad under various appellations, *e.g.*, dysfunction of the gall-bladder, kinks of the cystic duct, strictures of the ureter, and the like. In both the urinary and the biliary tract not infrequently typical colics take place simulating attacks of lithiasis and most careful study and most searching operative investigation fail to find a stone or any evidence of acute inflammation. In some cases these attacks occur after removal of the gall-bladder and cannot be ascribed to adhesions or to the abdominal wall giving away. Aschoff says we must now recognize that the occurrence of dyskinetic attacks of cramp in the gall-bladder region are more frequent than was formerly thought.

The study of some of these cases in the urinary tract has proven of great interest and it is possible that an analogous explanation may apply to the somewhat similar picture in the urinary tract. The urine is a complex

solution containing many ingredients in solution. Recent work has suggested that many of these are in a supersaturated solution by virtue of the action of the colloids. Pauli and Samic similarly showed that calcium phosphate and calcium carbonate in albuminous solutions are seven times more soluble than in water. If then a change in the colloidal system takes place the salts (*i.e.*, crystals) will, naturally, be thrown out in the urinary passages and what is called a colic due to a " shower " of uric acid crystals passing down the ureter, will occur. On voiding such urine the crystals will be in solution again, provided the colloid is reversible, and if allowed to stand, in the course of a day or two the nebecula (colloid) separates and the uric acid crystals are again apparent as crystals. The process inside the body is probably an instantaneous one—totally dissimilar from that seen in the test-tube. I have seen this instantaneous change in urines as they have passed through the female urethra; the patient passing a white opaque urine due to sudden dropping out of phosphates while the residual urine immediately obtained by catheter was yellow and clear. Similarly, in the upper tract, I have apparently induced a colloidal change by passing a ureter catheter and induced a unilateral fresh phosphaturia, and from the second side obtained clear urine such as was found originally in the bladder.

These phenomena have as yet been studied too little, and to apply the same methods to the biliary tract has been impossible. The presence of numbers of cholesterin crystals in the duodenal secretion or in the gall-bladder in colic cases in which no stone or inflammation was found, might establish the existence of a process analogous to that seen in the urinary tract. But even without this evidence, the therapeutic test offered by a change in diet suggests that similar colloidal disturbances are present in both systems, and that the over-concentrated solutions of uric acid in the one and of cholesterol in the other, are at fault. Restriction in protein intake controls the attacks of uric acid showers, and restriction in fats seems to have a similar effect in many of these biliary cases.

Lithiasis.—Turning to the question of lithiasis or stone production in these tracts, despite our imperfect knowledge, striking analogies are readily discerned. For many years the teachings of Naunyn held the field and gall-stones were considered the result of the combined activities of bacterial infection and stagnation of bile. In a study of these published some twenty years ago, it appeared to me that possibly a third factor—which might be a disturbed hepatic metabolism, was an essential element. Since then Aschoff has rather successfully separated the pure radial cholesterin stones from the whole group and emphasized the fact that they owe their origin to a faulty metabolism of the liver. He again called attention to the fact that these stones are usually found in bladders in which there is no sign of infection, or of past or present inflammation. Apparently the cholesterin is sedimented in the slowly moving bile of the gall-bladder and the crystalline stone forms. In the

urinary bladder of prostatics, previous to the period of infection, a very similar sedimentation is favored in the residual urine, and in these cases we often encounter uratic stones. In both cases, possibly a temporary spilling over of the stone material, an increased excretion, may contribute to produce the colloidal instability that leads to the precipitation of the stones. In the kidney such primary stones are more difficult to study—but here in the kidney pelvis and calyces are encountered uric acid, oxalate, cystin, Xanthin and indigo stones which have appeared to develop without the interference of infective agents, possibly due to processes analogous to those underlying the formation of the pure cholesterin stones. The analogy between the uric acid (and uratic) stones and the cholesterin stones is very striking. Owing to the obscurity of the metabolic processes underlying the other types of renal stones just mentioned, one must hesitate in grouping them together too intimately.

When, however, infection sets in, a totally different type of stone develops and these secondary stones are quite typical. In the biliary tract we find the cholesterin pigment calcium stones, and in the urinary tract (both in the kidney and in the bladder) the various more or less hard phosphatic (ammonium magnesium phosphate and calcium magnesium phosphate) stones. These may contain a nucleus of the primary type and are usually built up in layers and have a network of proteid material derived from the inflammatory exudate in which they developed. Thus it is apparent that there is a striking analogy in the formation of the primary as well as of the secondary post-infection stones in both the biliary and the urinary tracts.

Infections.—In studying the behavior of these two extensive excretory systems, it is quite striking to see how readily the bacteria (toxins?) are passed through these glands without any apparent damage to their parenchyma. For many years it has been known that in bacteriæmias the offending organisms are excreted in the urine. Apparently a very similar process takes place in the liver and biliary system, for under such circumstances in the bile a great variety of bacteria have been recovered. This transit through these two systems seems to do no permanent damage unless foreign bodies or obstructive conditions obtain and then local inflammatory changes may be induced.

In the kidneys bacillary and coccic infections are very common and many of these produce such mild local symptoms that they are liable to be overlooked. Many such infections have been misinterpreted. Undoubtedly in the majority of the cases an almost complete repair and restoration of function occurs. Do analogous infections occur in the biliary system? Some writers believe infections of the liver are "extraordinarily" common (Naunyn, Poppert, Aschoff). If they are common, owing to the lack of local symptoms and laboratory evidence, we are surely not recognizing them in the clinic, and further attention must be directed towards their diagnosis. Undoubtedly, the liver has marvellous recuperative power and being a remarkably silent organ,

many a febrile disease may be misinterpreted when the pathology lies within this organ.

The repeated attacks of infection in the kidney parenchyma eventually lead to the peculiar scarred kidney of old pyelonephritics, and if similar processes occur in the liver, one cannot but wonder whether some of the changes of the cirrhotic liver are not developed in this same way.

In closing this very inadequate study of some of the more striking similarities between the biliary and urinary tracts, one cannot but feel apologetic for attempting to put in words much that is still in the developmental stage. The parallelism has impressed me as too striking to be passed by, and I am sure it will stimulate further work and lead to a clearer understanding.

AN ECTOPIC (PELVIC) COMPLETELY FUSED (CAKE) KIDNEY ASSOCIATED WITH VARIOUS ANOMALIES OF THE ABDOMINAL VISCERA

By William W. Looney, M.D.
and
Durwood L. Dodd, M.D.
of Dalias, Texas
Department of Anatomy, Baylor University College of Medicine

During the dissection of a male cadaver, about seventy years of age, many anomalies of the abdominal and pelvic viscera were found which, from the embryological and clinical standpoint, make the condition of more than passing interest.

The jejunum, ileum, and ascending and transverse cola were found to be suspended by a common mesentery, resembling very closely the mesentery of the fœtus after rotation of the gut and before descent of the cæcum. The root of the mesentery, instead of having a basal attachment of five inches or more, was attached for about two inches around the superior mesentery artery (Fig. 1). The duodenum possessed a short mesentery almost continuous with that of the jejunum.

After the removal of the intestines and the peritoneum from the posterior abdominal wall, both lumbar regions were searched for the kidneys. Having failed to find them in their normal position, our attention was directed to a rather large mass lying over the right sacro-iliac joint and extending into the pelvis which proved to be a completely fused (cake) kidney, nearly circular in outline, and possessing two distinct ureters, each measuring about five inches in length, and opening normally into the bladder. The anterior or ventral surface of the kidney showed marked lobulation but no indication of a separation into right and left portions. The ureters arose separately from this anterior or ventral surface from four extrarenal calyces, which united about one inch from the kidney. The posterior surface of the kidney was perfectly smooth and concave.

Further search failed to reveal a suprarenal gland on the right side, although a perfectly normal one was found in its normal position on the left side.

The left testicle was perfectly normal, both as to development and descent. The right testicle was lodged against the kidney in the right iliac fossa. The inguinal canal was examined and found to contain the processus vaginalis extending all the way from the abdominal (internal) inguinal ring to the bottom of the scrotum. Below the subcutaneous (external) inguinal ring this vaginal process was obliterated. The portion within the canal was patent and contained a small peritoneal sac, which communicated with the peritoneal cavity. A loop of the vas deferens was found throughout the entire length of the inguinal canal and behind the vaginal process. The proximal part of the loop was obliterated from its most distal point up to the testicle, although the distal portion was open and extended back up through the inguinal canal and downward and medially to the base of the prostate. The penis was rather infantile in character.

The kidney received its blood supply from three larger and several smaller arteries. The left renal artery arose from the lower end of the left common iliac artery while the middle renal artery came from the angle of bifurcation of the aorta. The right renal artery arose from the proximal part of the right common iliac artery. One large renal vein, composed of several tributaries, passed out of the anterior surface of the kidney and joined the lower end of the vena cava. Several smaller veins came from different portions of the kidney and joined the common iliac veins.

In all probability the fused type of kidney—either horseshoe, pancake or bean-shaped—is the result of failure in differentiation of the two primary mesenchymal masses from which the kidneys are developed.

Reports of completely fused kidneys are met with in the literature; but the frequency of this occurrence is not definitely known, as authors differ

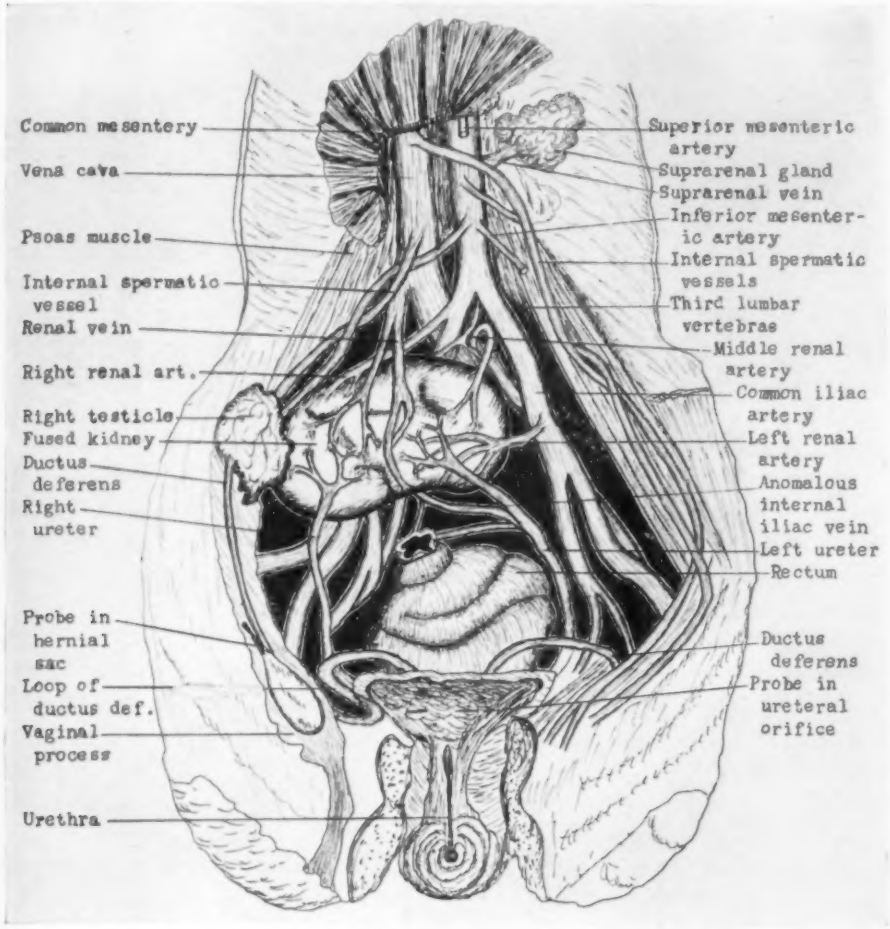


FIG. 1.—The intestines and a portion of the mesentery have been removed to show the other viscera. The ventral wall of the vaginal process has been dissected away to show the contained hernial sac. The anterior walls of the bladder and urethra have been cut away and the left testicle removed.

considerably in their tables of percentages. Lipshutz and Hoffman [1] state that the per cent. of fused kidneys is about 1 in 671. This figure is probably too high.

We are unable to offer any definite explanation as to why one or both kidneys may fail to ascend and remain in or near the pelvis. A number of different varieties of such kidneys are reported in the literature, among the most interesting of which are the cases of Cullen [2] and Polk.[3] These authors fail to offer any particular reasons for the occurrence of pelvic kidneys; but,

in all probability, the condition is purely accidental, resulting from some mechanical interference. We are not certain why kidneys ascend at all. It is obvious, however, that the kidney in the case herein reported was never any higher than its present position, since the ureters are short, and since its blood supply is derived from the arteries in the immediate locality.

The absence of one suprarenal gland is rather infrequent; and while we cannot state with absolute certainty that the right suprarenal gland did not develop in our specimen, we feel reasonably sure that such was the case, since no trace of a degenerated structure was found. A few reports of one suprarenal gland are to be found in recent articles dealing with this subject; and their absence is usually associated with single or fused kidneys, as reported by Abell [4] and others. It is no wonder that one or the other of these structures should fail to develop, for we may recall that the cortical substance of this gland appears in the cephalic end of the more or less undifferentiated mesonephric mesothelial tissue. As this tissue exists only for a while and then undergoes degeneration, it is reasonable to suppose that the cortex of the suprarenal gland also disappears in some instances. Also, the cortical substance may develop in isolated masses and its identity be lost as a distinct gland. The medullary substance of this gland is subject to many variations, since it is supposed to be of the same origin as the sympathetic ganglia.

It is uncertain just why the vaginal process in this specimen should have descended completely, as the testicle never proceeded any lower than the iliac fossa. However, we may recall that this process begins to push through the abdominal wall at about the third month of fœtal life; and, at a corresponding period, the testicle is in the iliac fossa.

We are unable to account for the partial descent of the loop of ductus deferens; nevertheless, the condition bears a close relation to sliding herniæ (usually of the cæcum and appendix) accompanying undescended testis. Eisendrath [5] states that sliding herniæ usually occur only in cases similar to the one described. In this connection we might state that we have seen two sliding herniæ of the sigmoid colon in individuals with normally descended testicles.

Cullen [6] reports an interesting case in a female whose round ligament had descended into the inguinal canal very much in the same manner as the ductus deferens had done in our specimen.

REFERENCES

[1] Lipshutz and Hoffman: A Contribution to the Knowledge of Fused Kidneys. Annals of Surgery, vol. lxviii, No. 1, p. 39, July, 1918.

[2] Cullen: A Right Pelvic Kidney; Absence of the Left; Absence of the Uterus; Both Ovaries in the Inguinal Canal. Surg., Gyn. and Obst., vol. xi, No. 1, p. 73, 1910.

[3] Polk, 1882, Cited by Glazebrook: N. Y. M. J., July 22, 1905.

[4] Abell, Irwin: Single Kidney to Left of Sacro-iliac Joint, no Left Suprarenal and One Ureter. Surg., Gyn. and Obst., vol. xxiii, p. 33, 1916.

[5] Eisendrath, Daniel N.: Undescended Testis. Annals of Surgery, vol. lxiv, No. 3, p. 324, September, 1916.

[6] Cullen: (See 2).

RENAL ARTERIAL VARIATIONS AND EXTRAPERITONEAL ABDOMINAL NEPHRECTOMY*

By Benjamin Lipshutz, M.D.

of Philadelphia, Pa.

surgeon to the mt. sinai hospital

and

Clarence Hoffman, M.D.

associate in anatomy of the jefferson medical college

from the daniel baugh institute of anatomy

Departures from the conventional type in anatomic structures are slowly assuming a place of importance in the practical field. All that the observant student has to do is to witness the dissection of a large series of cadavers to have impressed upon him that there is no fixed and unalterable anatomic type in very many of the parts of the human body. Fortunately, the wide use of röntgenology has come to the aid of the physician and surgeon in the delineation and determination of anatomic conformation.

The unique manner of the development of the kidneys which consists in the succession of functional kidneys, particularly predisposes them to a wide range of variations and anomalies. Additionally, the widespread practice of pyelography and röntgenographic studies of the kidneys advances the variations in the anatomy of the kidney to a plane of considerable diagnostic importance. A review of the surgery of the kidney, particularly the complications of nephrectomy further emphasizes the fact that renal anomalies are a matter of grave concern to the surgeon.

Description of Specimen.—The specimen here reported is unusual in several respects: 1. The marked vascularity of both kidneys, illustrating in a composite form the important variations of the renal arteries and veins. 2. Bilateral absence of the ventral lip of the hilus, and 3. Bilateral hypertrophy and moderate ectopia.

Both kidneys are larger than normal, the right being somewhat smaller than the left. In shape the kidneys are altered, the right being oval and more closely approximating in shape a normal kidney, the left elongated and flattened. On both kidneys the hilus is on the ventral surface. The anterior (ventral) lip of the hilus, as in the horseshoe or fused kidney, is absent and is represented as a low ridge, curving lateralward to reach the inferior pole on the ventral surface of the kidney. The normal sinus renalis is also absent, the vessels, nerves and calyces penetrating a broad, convex surface continuous with the ventral surface of the kidney, as in the case reported by Harvey. The posterior lip of the hilus is absent.

Blood-vessels (Fig. 1): *Right Kidney.*—The right kidney receives its blood supply from three large renal arteries. The most cephalic of the latter arises separately from the ventrolateral aspect of the aorta immediately caudal to the origin of the superior mesenteric artery. This renal artery divides into two large branches before entering the cephalic extremity of the kidney at its medial border. The artery lies dorsal to the renal veins and cephalad to the pelvis. The second renal artery similarly arises from the aorta, crosses the ventral aspect of the vena cava inferior and penetrates the median

* Read before the Philadelphia Academy of Surgery, May 3, 1926.

border of the kidney unbranched. It lies in front of the renal vein and pelvis and gives off the internal spermatic artery.

The third renal artery arises from the bifurcation of the aorta immediately to the right of the middle sacral artery. It breaks up into branches before entering the inferior pole of the kidney.

A normal right *renal vein* arises by four large radicles from the ventral surface of

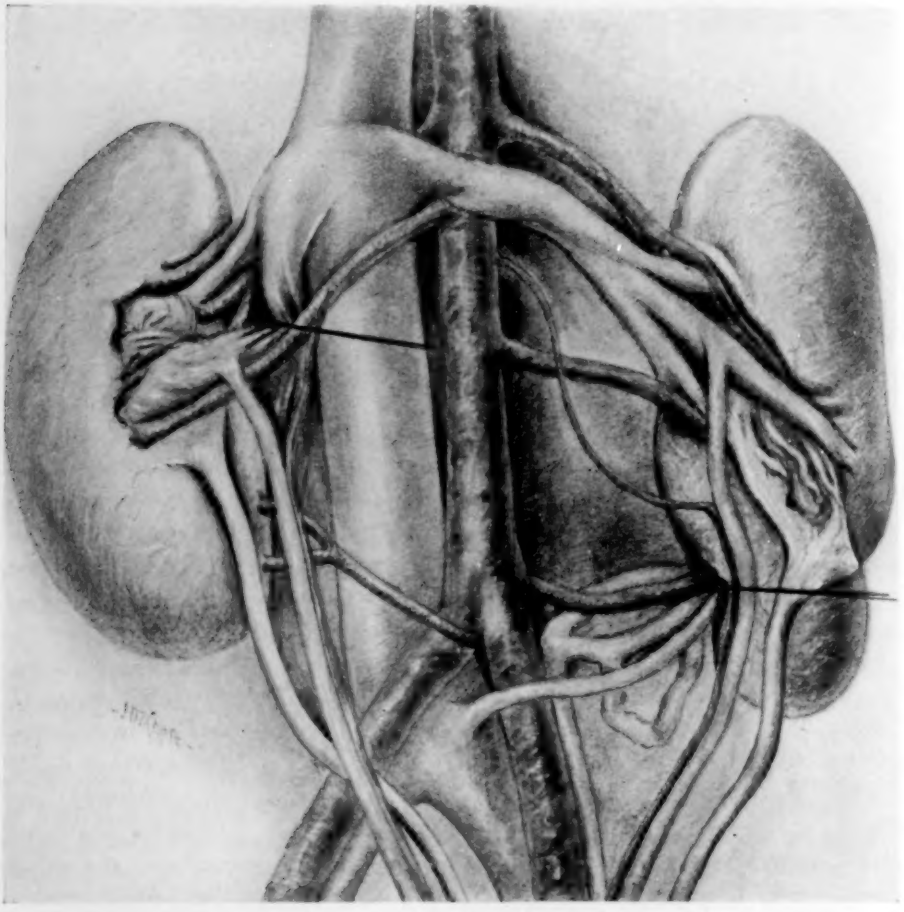


FIG. 1.—For description see text page 525.

the kidney and receives the internal spermatic vein. The vein is ventral to the artery and its radicles embrace the branches of the most cephalic renal artery.

Left Kidney.—The left kidney also receives its blood supply from three renal arteries. The most cephalic renal artery arises separately from the aorta and bifurcates into two large branches about 2 cm. from the medial border of the cephalic pole of the kidney, before it penetrates the renal parenchyma. It lies cephalic to the renal vein. The middle renal artery, likewise, arises as a separate branch of the aorta, passes dorsal to the pelvis and is unbranched as it enters the kidney substance, 1 cm. from the median border. The most caudal renal artery arises from the aorta, courses ventral to the inferior renal vein, dorsal to the pelvis and is distributed to the caudal pole of the kidney.

Three large renal veins emerge from the left kidney. The most cephalic is the largest and arises by four radicals from the ventral surface of the kidney, crosses the

aorta and enters the lateral aspect vena cava inferior. This renal vein receives the internal spermatic vein. The two caudal veins assume a rather bizarre formation, as pictured in Fig. 1. The second renal vein which grooves and encircles the lateral border of the kidney, arises by several radicles and emerges from the caudal pole of the kidney near the lateral border. The latter vessel passes ventral to the common iliac artery to enter the vena cava inferior just above its bifurcation. The most caudal renal vein is very unusual. It arises from three radicles which have anastomotic venous chains connecting one to the other, the vessel then pursues a course lateral to the common iliac vein and enters the latter just before it bifurcates.

The accessory renal veins as noted in this specimen, show a considerable range of variations. Variations in the renal veins are more frequently encountered than on the arterial side.

Unilateral absence of the anterior lip of the hilus is not uncommon. In a series of 80 cadavers during the past year, this variation was observed three times. Bilateral absence of the anterior lip of the hilus is exceedingly rare, the specimen here reported being the only instance encountered in over 1000 cadavers.

The ventral lip is usually found absent in fused or horseshoe kidneys as noted by the writers in 1918. The kidney during its growth undergoes a migration and rotation about its axis. The displacement of the hilus from its mesial border to the ventral surface shows the undeveloped character of the labia (Harvey). This specimen with its undeveloped labia of the hilus, excessive vascularity, moderate ptosis and in the arrangement of its calyces resembles closely in its anatomic formations a fused or horseshoe kidney.

Anomalous Renal Blood-vessels.—Anomalous renal blood-vessels entirely apart of their interest to the student of embryology and morphology are also of a considerable significance from a clinical and surgical viewpoint. Variation in the number and distribution of the renal arteries are perhaps more frequently met with than any other of the larger arterial trunks and the commonest variation is the presence of an additional renal artery. The vessels vary in number, in origin, and in their anatomic course and relations. As many as five or six to one kidney have been reported. In many instances the presence of accessory or multiple renal arteries is associated with an arrested development in the kidney and occasionally of the ureter. A fact which suggests that multiple renal arteries are produced by some deviation from the normal development in the vessels of the kidney. Abnormalities in the form and position of the kidneys in association with accessory renal arteries have been pointed out by a number of observers. In general the kidney deviates from its normal reniform shape in proportion to the number of vessels. Rupert in 35 of 50 cadavers found variations in the renal arteries without any change in the normal position of the kidneys, although their shapes were altered. In horseshoe kidneys, as noted by the writers in 1918, additional renal arteries are the rule rather than the exception.

Organs which make extensive migrations from one position to another, may retain vessels from their original position or receive or incorporate vessels of the new region invaded. The kidney during its growth migrates and undergoes a rotation around its long axis. The instances of accessory renal arteries arising from iliac arteries from the middle sacral and inferior mesenteric arteries are to be considered as persisting embryonic vessels of

the capillary plexus supplying the normal embryonic kidney. Developmentally, these arterial anomalies bear no relation to the normal adult renal artery, for the kidney does not receive the latter artery until it reaches its definitive position.

Concerning the range of frequency of accessory renal arteries, some discrepancy is noted in the relative proportions of upper and lower accessory renal polar arteries as reported by various observers. The accessory renal arteries may be distributed to either pole of the kidney. It is particularly noteworthy that an accessory superior or inferior polar renal artery may have its origin from the renal artery proper and not from the aorta. Eisendrath, in his study of the variation of the renal vessels, directs especial attention to the latter renal arterial variation. The presence of additional renal arteries which arise directly from the aorta, is too well known to merit further consideration. When it is recalled that some type of renal arterial variation is present in from 20 to 33⅓ per cent. of all subjects, the only safeguard in operations upon the kidney, is to be constantly on the lookout for them. Eisendrath additionally mentions the presence of variations in the retropelvic arteries and veins, which vessels may be injured in the operation of pyelotomy.

FIG. 2.—Hydronephrosis, the result of an accessory inferior polar renal artery. (Modified from Rumpel.)

Undoubtedly, insufficient attention has been directed to the variations in the renal veins which are subject to a greater range of variations than are the renal arteries and surgically are just as important, particularly in the performance of a nephrectomy.

Kinking or compression of the pelvis or of the ureteropelvic junction by

an accessory inferior polar renal artery, arising from the renal artery proper or independently from the aorta, may produce attacks of high obstruction of the urinary tract and lead to the development of a hydronephrosis. Hydronephrosis associated with aberrant renal arteries usually occurs in the young adult.

Figure 2 illustrates a definite hydronephrosis, the result of an aberrant renal artery in a kidney removed at operation by Rumpel. The patient was a boy of twelve who gave a clinical history of intermittent attacks of renal colic. The specimen shows clearly how an aberrant artery can mechanically interfere with the normal urinary current. The S-shape loop of the ureter is, however, a secondary change which followed the abnormal dilatation of the pelvis of the kidney.

Braasch, in discussing Foley's paper on "The Diagnosis of Anomalous Renal Artery," says that if the surgeons would stop and look carefully for the cause of hydronephrosis, the anomalous vessel would be found more frequently.

From a surgical standpoint, renal arterial and venous variations, emphasize the necessity of accurate anatomic visualization in operations upon the kidney.

Extraperitoneal Abdominal Nephrectomy.—During 1915 and 1916 a considerable number of dissections were performed in order to study the blood supply of the ureter. The ease and clearness with which the kidney can be exposed through an abdominal incision, when once the proper plane of peritoneal cleavage is established, was noted. Additional anatomic observations and dissections extending over a period of ten years, confirm this opinion.

The particular indications and advantages of abdominal extraperitoneal nephrectomy were discussed in a previous paper (February, 1925, ANNALS OF SURGERY). The more important complications and dangers attendant upon the removal of the kidney are here considered. It is our belief that the anatomic hazards of nephrectomy can be largely eliminated through the utilization of ventral nephrectomy. A review of the surgery of the kidney and of the complications of nephrectomy, discloses that injury of the posterior portion of the parietal peritoneum which is in intimate contact with the ventral surface of the kidney, is the most common accident during the performance of a nephrectomy. Immediately upon its occurrence, suture of the peritoneum is indicated. The possibility of a tear of the parietal peritoneum, from our anatomic observations, is considerably lessened when the kidney is exposed by means of the extraperitoneal abdominal route.

Here and there scattered sporadically through the literature are reports of injury to the cæcum, colon descendens or colon ascendens with resulting fecal fistula following a nephrectomy.

The anatomic intimacy of the different divisions of the colon to the ventral surface of the kidneys, particularly the nearness of the colon descendens and left colic artery to the ventral surface of the left kidney, are

important surgical relations. Even in cadaver specimens the greatest possible care is occasionally necessary in the separation and detachment of the parietal peritoneum with the colon descendens from the ventral surface of the left kidney in order to avoid injuring the peritoneum and its related structures. With the ventral exposure, the colon is under direct vision during every mechanical manœuvre and once the plane of peritoneal cleavage is established, the detachment of the peritoneum is easy and safe.

Hemorrhage from an overlooked accessory renal artery, or from an aberrant renal vein, either immediate or post-operative, may be followed by a fatal result. Hemorrhage from a normal renal artery may follow when the kidney pedicle is short. The isolation of the pelvis or ureter may occasionally be attended by hemorrhage due to the variations of the dorsal retropelvic arteries and veins, unless a careful anatomic exposure is obtained. One of the dangers of the so-called subcapsular nephrectomy is the possibility of completely overlooking an accessory renal artery which arises from the aorta or from the renal artery proper.

Among the most hazardous and disconcerting complications of nephrectomy is a tear or rupture of the vena cava inferior. The relative shortness of the right renal vein which occasionally adds to the difficulties of a right-sided nephrectomy, is the most frequent cause of this accident. Injury to the vena cava inferior is particularly liable to occur during a nephrectomy for malignancy. Fonstein has collected 75 cases in which this perilous complication has occurred. Altogether there were 22 deaths, a mortality of 29 per cent. It is probable, however, that this accident occurs more frequently than reports indicate and in all likelihood is attended with a higher mortality than that noted above (29 per cent.). Suture of the lateral wall apparently gave the lowest mortality, as a method of treatment, and tamponade of the wound, the highest. Here again, abdominal nephrectomy with the clear visualization of the pedicle of the kidney and of the vena cava inferior insures against injury of the latter vessel.

Extraperitoneal abdominal nephrectomy further eliminates the occasional exigency where it becomes necessary to leave some form of clamp applied to the renal pedicle for a period of 48 to 72 hours. Aside from the danger of secondary hemorrhage, on removal of the clamps, a number of cases of duodenal fistulæ have been reported as the result of their use. Instances of injury to the diaphragm and pleura are also reported following the application of clamp to the renal pedicle for a period of 48 to 72 hours.

When properly carried out the extraperitoneal abdominal approach for exposure of the kidney gives a splendid and clear view of the kidney and its pedicle. The vessels of the kidney can be secured without disturbing the kidney from its bed. Separation of the parietal peritoneum is effected on a broad, clear surface and under direct vision. The use of lateral semi-flexed position is avoided. Furthermore, as noted, it eliminates the danger of injury to accessory renal arteries, veins, the post cava, the duodenum and colon.

The difficulty of delivering a large kidney or a kidney with a short pedicle is greatly reduced.

This method of nephrectomy finds its greatest field of usefulness in the removal of large cysts or tumors of the kidney. The surgeon is able to carefully examine the kidney *in situ* and determine the type of tumor or cyst. Rehn has been impressed by the discrepancy between the rarity of metastases from an untreated hypernephroma and their extreme frequency after radical operations. He attributes this to the rough handling of the kidney during the operation. He states that metastases can be avoided by exposing the renal vein first, instead of last and throwing a provisional ligature around it before drawing out the kidney.

Anatomically, the abdominal extraperitoneal exposure of the kidney area permits the surgeon to ligate and divide the renal pedicle as the first step of the nephrectomy, with the kidney lying undisturbed in its natural bed. With this technic, metastases through the renal vein, incident to the trauma of separating and delivering the kidney, is avoided.

Congenital lesions of the kidney, such as horseshoe kidney with its variations in blood supply and other abnormalities, are anatomically more accessible through the abdominal approach. In traumatic lesions which involve the abdomen and kidney or kidney area, the abdominal cavity and the retroperitoneal region can be explored through the one incision. Recently one of us (Lipshutz) encountered two cases of so-called massive hemorrhages of the renal bed, the result of automobile accidents. In both instances the evidence of hemorrhage and coincident shock with a tense, rigid abdomen, and apparently negative evidence of injury to the kidney and thorax, prompted an immediate abdominal operation.

The abdominal exploration was negative, but appearing on the posterior parietal peritoneum was a large bluish tense area. The peritoneum was then closed and the parietal peritoneum separated from its mural attachment to gain an exposure to the retroperitoneal region. A massive hemorrhage of the renal bed was present in both cases. The bleeding was controlled by hot packs and a number of ligatures. The abdomen was closed in layers and a small pack was placed in the renal bed which was brought out through a stab wound in the loin. Recovery was smooth and uneventful in both instances.

Like any other operation its value must be measured by the yardstick of practical experience and clinical end results. In no other way can it acquire any semblance of permanency. Anatomically, the operation rests upon a firm basis, for it permits a clear exposure of the kidney that can be carried out with rapidity and safety. There can be no objections to this operation on the grounds of anatomy. While our practical experience with this operation is, as yet, limited to but a small number of cases, it has proved satisfactory in every way.

Thanks are due to Dr. J. Parsons Schaeffer, head of the Department of

Anatomy, for permission to report these specimens and for his generosity in placing at our disposal all of the available anatomical material.

REFERENCES

Bremer: American Jour. Anatomy, vol. xviii, p. 179, September, 1915.
Eisendrath: ANNALS OF SURGERY, June, 1920.
Fonstein: Zeitschrift fur Urol. Chir., p. 727, vol. xvi, 1924, p. 50.
Harvey: Anatomical Record, vol. viii, p. 333.
Lipshutz and Hoffman: ANNALS OF SURGERY, July, 1918, p. 39.
Lipshutz: ANNALS OF SURGERY, February, 1925.
Rehn: Zeitschrift fur Urologie, vol. xix, pp. 5, 74, 1925, p. 38.
Rumpel: Beiträge Zür. Klinisch Chir., vol. cxxvi, p. 1, 1922.

URINARY OBSTRUCTIONS IN CHILDHOOD*

By Charles G. Mixter, M.D.

of Boston, Mass.

From the Surgical Service of the Children's Hospital of Boston, Mass.

Urinary obstruction is unconsciously associated with the mental picture of the later years of life. One visualizes the old prostatic with the distressing train of symptoms following back pressure and diminished renal function.

Causes of Urinary Obstruction in Childhood.

			No. of cases
A. Intrinsic lesions of urinary tract.	1. At uretero-pelvic junction.	a. Stricture uretero-pelvic junction	7
		b. Faulty insertion ureter into pelvis (with hydronephrosis)	2
		c. Kinks of ureter (with hydronephrosis).	6
	2. At vesical insertion.	a. Stricture of ureteral orifice.	3
		b. Cystic dilatation of orifice.	2
	3. In posterior urethra.	a. Congenital stricture.	2
		b. Valves at veramontanum.	6
		c. Hypertrophy of veramontanum	0
B. Condition producing obstruction by pressure from without.	1. Accessory renal vessels.		6
	2. Reduplication of pelvis and ureter.		7
C. Stasis produced by primary dilatation of ureter. No demonstrable obstruction.	1. Megalo-ureter.		5

Fig. 1.

This clinical picture is well understood and adequately handled surgically. Obstruction of the urinary outflow in infancy and early childhood, on the other hand, has rarely been recognized except at post-mortem examination. The frequency of its occurrence and its menace to health and life in early years has not been appreciated.

Urinary stasis is sooner or later followed by infection. Unless the obstruction is relieved and adequate drainage established, it is practically

* Read before the American Surgical Association, May 26, 1926.

impossible to eradicate the infection and ultimately one or both kidneys are destroyed by pyelonephritis. The importance of the underlying factor of urinary obstruction in childhood is little realized and many of the cases are seen only when the destructive process is far advanced. A complete urological examination can and should be made at any age when signs of urinary retention are present or when there is persistent or recurrent pyuria.

Briefly the routine of examination has been as follows: After the usual urinary, renal function, and blood chemistry studies, a cystogram is made with 20 c.c. of 12 per cent. sodium iodide in both the antero-posterior and lateral positions to ascertain the presence of a ureteral reflux, dilatation of the posterior urethra, or diverticulum of the bladder. A cystoscope is then passed, both ureters are catheterized and pyelograms are made. The catheters are then withdrawn to the lower ureter and ureterograms are made. The ureters are then drained and a final film is taken. This will show whether there is delayed emptying of the pelvis or any portion of the ureter. The divided renal function test has been unsatisfactory as the catheters used are necessarily so small that there is urinary leakage around them, vitiating the test. Cystoscopy can usually be carried out in girls over eight or nine years of age and boys over eleven years, under local anæsthesia. In younger children a general anæsthetic is required.

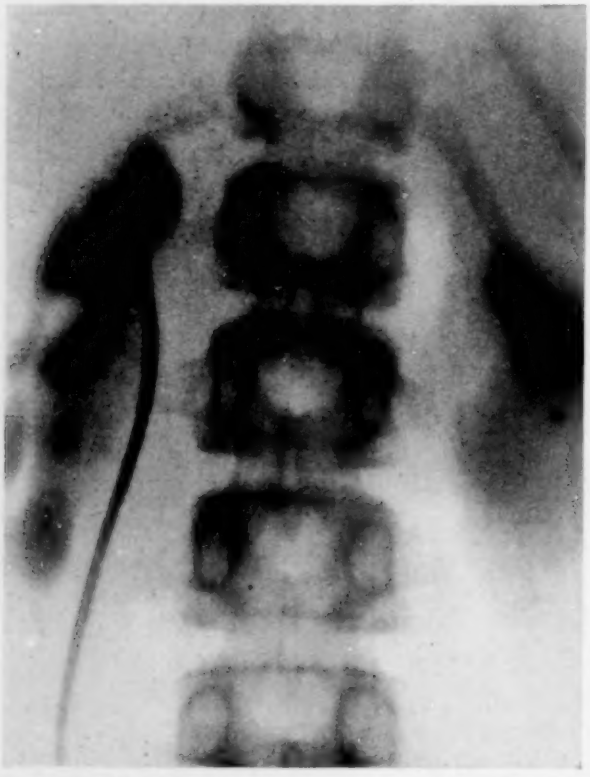


FIG. 2.—Faulty insertion at uretero-pelvic junction. No. 28860. Female, ten years, July 15, 1920. Uretero-pyelograms show advanced bilateral hydronephrosis. Greatly delayed emptying time of pelvis. Radiographic appearances suggest a faulty insertion of the ureter into the renal pelvis. Not confirmed by operation.

Usually children are referred for urological examination for one of four conditions: pyuria of long standing, acute attacks of abdominal pain of unexplained origin that may be referred to the kidney region, difficulty in urination and hæmaturia. The symptoms and physical signs of obstructive lesions are so indefinite that an accurate diagnosis is practically impossible without the aid of the cystoscope and uretero-pyelograms. Where vesical

retention is present, spina bifida occulta or other lesion interfering with bladder enervation, must be ruled out.

Leaving out of consideration phimosis and stricture of the external meatus, there are three regions of the urinary tract especially prone to

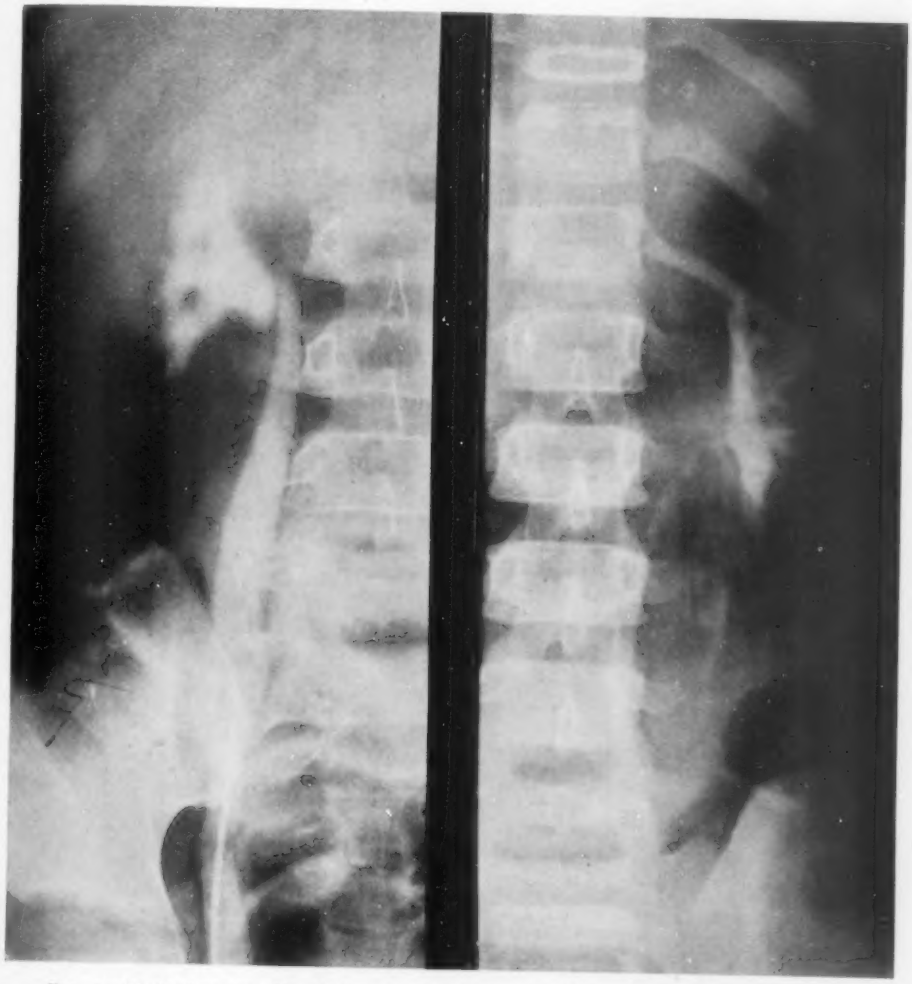


FIG. 3.—Kink of ureter. No. 88898. Female, eight years, November 17, 1925. Uretero-pyelogram shows marked hydro-ureteronephrosis on the right side, sharp kink at the uretero-pelvic junction. Cause of acute attacks of hydronephrosis. Exploration advised and refused.

obstructive lesions in early life, namely, the uretero-pelvic junction, the vesical insertion of the ureter and the region of the veramontanum in the male urethra. More commonly such lesions are congenital, rarely they may be acquired.

Stricture of the uretero-pelvic junction is followed by hydronephrosis. Inflammatory changes are usually found on microscopical examination of the sections taken through the point of stricture. Where sufficient renal tissue

is present in spite of prolonged back-pressure, a plastic on the uretero-pelvic junction is the operation of choice.

Congenital stenosis may occur, which in rare instances may be bilateral. Large cystic tumors are formed which can be identified by the thin shell of kidney substance spread out on the surface of the cyst. So little functional tissue is present, that nephrectomy is almost invariably indicated.

Faulty insertion of the ureter into the renal pelvis at too high a level will cause inadequate drainage. The greater the distention of the pelvis,

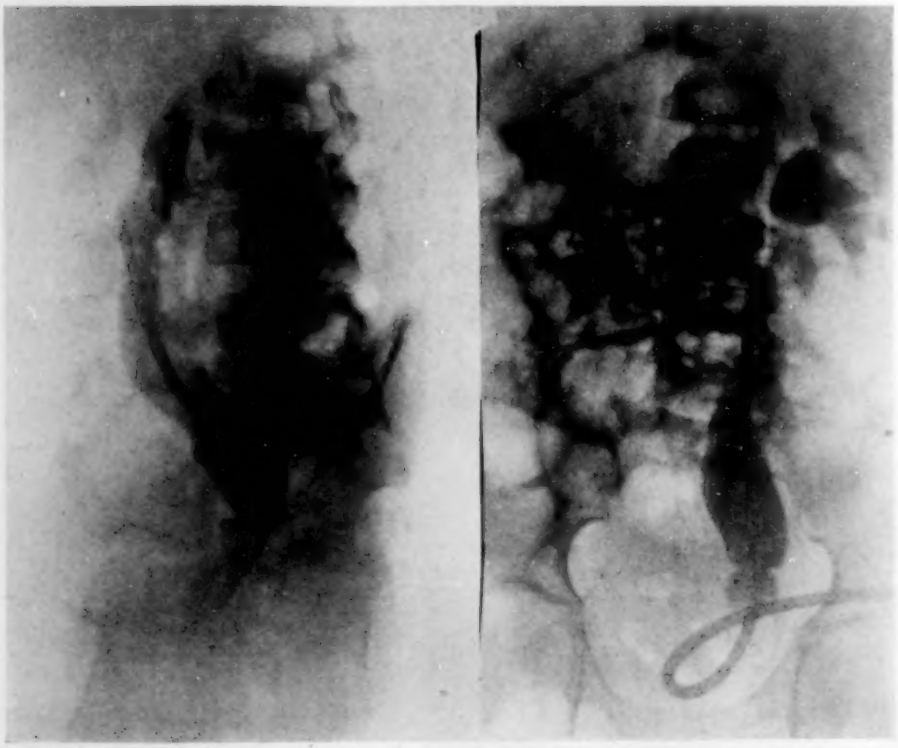


FIG. 4.—Stricture of ureteral orifice. No. 54441. Female, seven years. February 12, 1925. Post-operative radiogram. Before operation attempts to catheterize right ureter failed. Orifice would not admit the finest catheter. Cystotomy and plastic on right ureteral orifice with drainage. Tight stricture found at orifice causing attacks of hydronephrosis and dilatation of ureter.

the sharper the angulation at the uretero-pelvic junction and the more complete the obstruction. Where the hydronephrosis is slight and there is little evidence of renal destruction, a plastic on the uretero-pelvic junction, or section and reimplantation of the ureter into the most dependent portion of the pelvis, should be performed. Frequently, however, the kidney is so far destroyed by back pressure and infection, that the indication for nephrectomy is evident.

The ureter in early childhood is more tortuous than in the adult and ureteral kinks near the pelvic junction are common. It is difficult in any given case to determine just how far the angulation revealed by ureterogram may be responsible for urinary stasis and secondary infection. That a kink

can be the cause of urinary obstruction was clearly demonstrated in a recent case. Nephrectomy was done for a large hydronephrosis, the size of a grape fruit. The sharply kinked ureter was sectioned several inches below the pelvis. On removing the clamp the hydronephrotic sac remained dis-

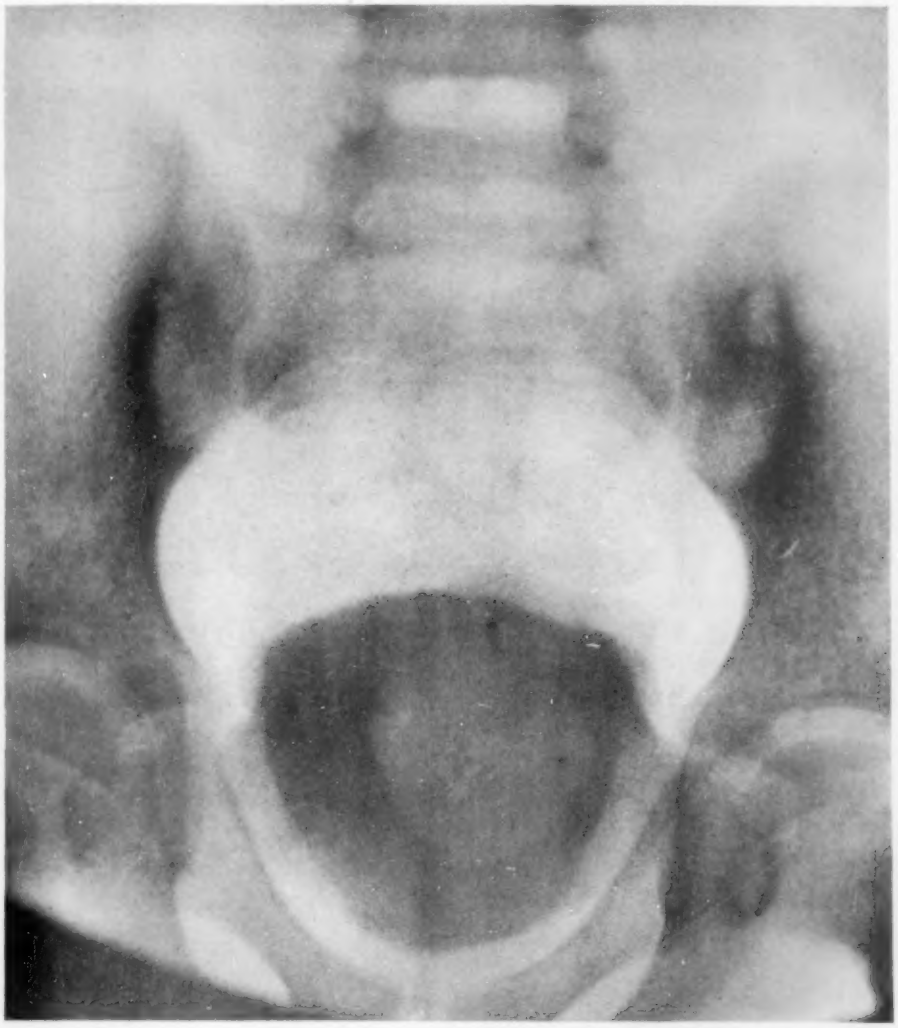


FIG. 5.—Cystic dilatation of ureteral orifice. No. 88255. Female, eight years, October 29, 1925. Child uræmic and died without surgical intervention. Autopsy specimen revealed a large cystic tumor at the site of the right ureteral orifice and a similar small cyst on the left. With a syringe and considerable pressure, no fluid could be forced through the stenosed orifices on the surface of the cysts after removal of the specimen.

tended, but by traction on the sectioned end of the ureter the angulations were eradicated, there was a free flow of urine and the distended pelvis rapidly collapsed.

At the vesical end of the ureter stricture of the orifice with or without cystic dilatation may occur. Stricture with cystic dilatation in one instance

was bilateral. In another patient, a boy of sixteen months, there was unilateral reduplication of the ureter and pelvis. One of the orifices was normal, the other emptied on the surface of a cystic dilatation. The cyst was of large size and involved the base of the bladder to such an extent that it occluded the internal urethral opening, producing vesical retention.

The intravesical partition of such cysts is thin, consisting of an external and internal lining of epithelium and an intermediate layer of connective tissue. Excision of this partition completely relieves the obstruction and is

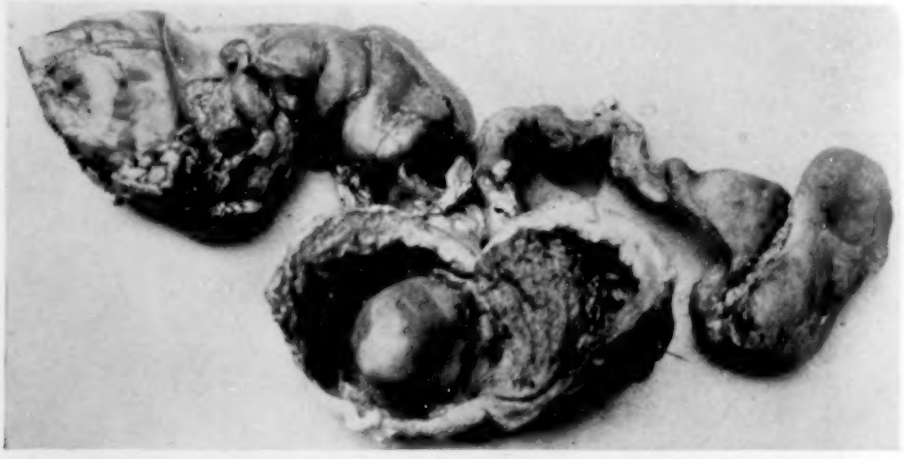


FIG. 6.—Autopsy specimen obtained from case referred to in Fig. 5, showing bilateral stricture of ureteric orifices with cystic dilatations. Bilateral hydro-ureteronephrosis.

more adequate than simply laying open the cyst, which procedure may be followed by cicatricial stenosis. Cystoscopic operative methods are impossible through the small calibrated instrument usually required for cystoscopy in childhood. Where fulguration can be employed, destruction of the vesical wall of the cyst can be accomplished satisfactorily by this means, if the dilatation is of small size.

Where stricture of the orifice is present, without cystic dilatation, slitting the intramural portion of the ureter from the opening upward for a distance of about one-half inch through a transvesical exposure, and drainage of the dilated and usually infected ureter with a good sized soft rubber catheter for ten days to two weeks, has secured a very satisfactory result.

Two views are held regarding the etiology of these strictures: first, that they are of congenital origin; secondly, that they occur as the result of infection. In view of the early age at which they may be encountered and the absence at times of evidence of marked infection on urinary examination, the former hypothesis would seem the more probable.

Diverticula may produce urinary retention from obstruction at the vesical neck. Situated usually in the vicinity of a ureteral orifice, they may dissect downward behind the base of the bladder as they enlarge until the distended sac shuts off the internal meatus. They are of rare occurrence in childhood

comprising approximately 5 per cent. of the total number of bladder diverticula reported and those causing retention constitute only a fraction of this small number. In our clinic for the past three years a routine cystogram has been taken in all cases of bladder retention or where the symptoms are those of difficulty in urination. No instance of diverticulum, however, has been encountered and none is found in the past records. Nevertheless it should be borne in mind as a possible cause of retention in childhood.

Obstruction in the male urethra occurs from congenital valve-like folds in the region of the veramontanum or from hypertrophy of the veramontanum itself. More rarely there may be a true congenital stricture. Recently fifty-six instances of valve formation have been collected from the literature and in 1923 seven examples of obstruction from hypertrophy of the veru were reported from the autopsy protocols of the Babies' Hospital in New York. These figures undoubtedly give a very inadequate idea of the frequency of occurrence of congenital urethral obstructions. The routine method of performing postmortem examinations does not include removal of the deep urethra with bladder and upper urinary tract. It is only in recent years that attention has been called to these lesions, since which time the number of cases observed in our clinic would suggest a higher incidence than these figures apparently indicate.

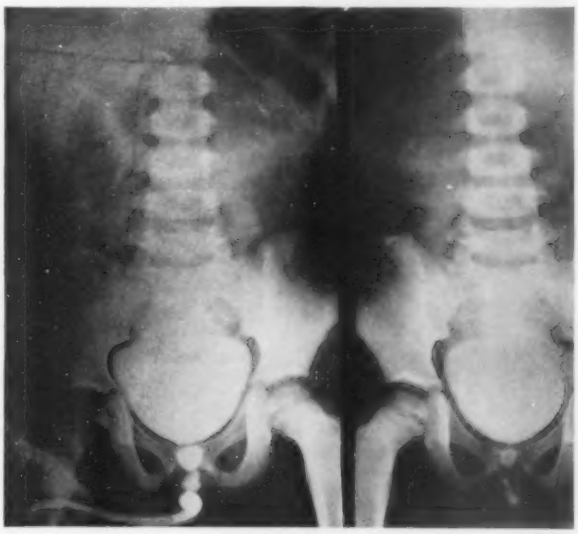


FIG. 7.—Congenital stricture of urethra. No. 45254. Male, six years, January 14, 1925. Cystogram shows a dilated posterior urethra. The distal urethra is outlined. The intermediate area is bounded distally by the strictured portion of the anomalous urethra and proximally by valves at the level of the veramontanum. Confirmed by perineal section.

Two instances of congenital stricture of the deep urethra have come under observation. This anomalous condition arises from failure of the downward growing proximal portion of the urethra to unite accurately with the inward growing distal segment during development. In the cases observed each segment ended blindly, save for a minute opening connecting them laterally, where the blind ends overlapped. The condition was clearly demonstrated by open perineal operation in one instance; in the other a coudé filiform passed the stricture and was followed by gradual dilatation with relief of bladder distention and marked general improvement.

The pathology produced in the urinary tract above the obstruction is striking. The bladder wall is greatly thickened with hypertrophy of the

musculature causing trabeculation, such as is seen in the cystoscopic examination of the prostatic bladder. The ureters are tortuous and dilated and there is bilateral hydronephrosis. The uretero-vesical sphincters are frequently incompetent, permitting a ureteral reflux to the kidneys, and a simple cystogram will often give a satisfactory uretero-pyelogram.

Removal of the obstruction may be accomplished by fulguration of the valve or hypertrophied veramontanum. In infants this procedure is not possible and usually in childhood suprapubic cystotomy or perineal section with excision is necessary.

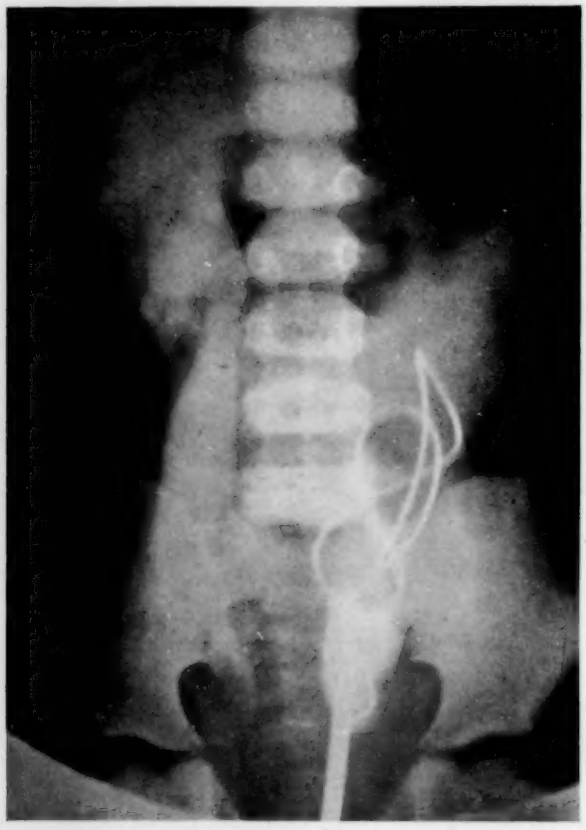


FIG. 8.—Urethral obstruction from congenital valves. No. 68121. Male, seven years, February 28, 1924. Uretero-pyelograms show enormous bilateral hydro-ureteronephrosis. Bladder trabeculated. A valve type of obstruction was demonstrated in the posterior urethra by suprapubic cystostomy.

In addition to the intrinsic obstructive lesions of the urinary tract in childhood, there are encountered two congenital anomalies producing urinary stasis by pressure on the ureter from without. These are accessory vessels running to the lower pole of the kidney and reduplication of the renal pelvis and ureter.

Accessory renal vessels may be either artery, vein, or both, and commonly the artery arises from the aorta and the vein empties into the cava. The vessel crosses the ureter at right angles near the uretero-pelvic junction and even slight mobility of the kidney may angulate and obstruct this point. This type of anomaly is not uncommonly bilateral. Should a case having typical renal colic from intermittent hydronephrosis disclose at operation an accessory lower polar vessel as the cause, the opposite kidney should be carefully studied. Even if there had been as yet no symptoms referable to this kidney, should the pyelograms demonstrate a delayed emptying time either in the prone or upright position, or should beginning hydronephrosis be evidenced by slight bulging of the inferior concave outline of the pelvis, exploration of this kidney should be seriously considered.

In the very early stage of intermittent hydronephrosis before the normal

elasticity of the renal pelvis is lost, the pyelogram will show no deviation from the normal outline. A girl of eight years was admitted to the hospital with the history of three attacks of right-sided abdominal pain during the preceding six months. The story and clinical findings were not characteristic of appendicitis. The urine was negative. Ureteral catheterization revealed nothing abnormal, though there was incomplete filling of the right pelvis. There was no distention of the pelvis and the calyces were not clubbed. Operation did not seem indicated, and while the child was awaiting her discharge from the hospital, there was an acute attack with severe pain, chill, fever, vomiting and a tense palpable tumor in the right kidney region. This mass disappeared within four hours. On exposure the kidney and pelvis appeared normal but there was an accessory lower polar vessel. This was tied and sectioned and in the two years since operation there has been no recurrence of the attacks.

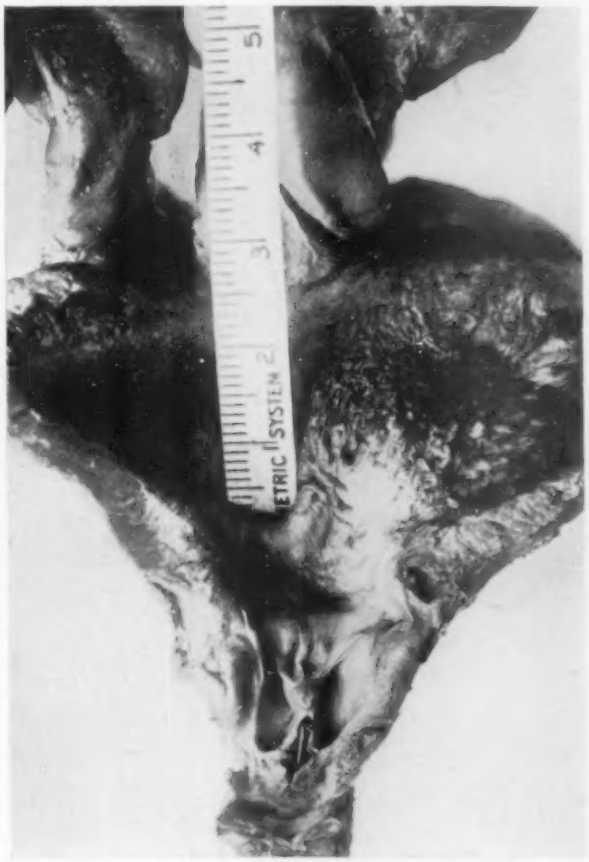


FIG. 9.—Urethral obstruction from congenital valve formation. I. H. No. 10593. Male, six weeks, April 26, 1923. Autopsy specimen shows obstructing valves running to veramontanum. Effects of back pressure are seen in the thickened trabeculated bladder, the tortuous, dilated ureters and the bilateral hydronephrosis with renal destruction.

Section of the accessory renal vessel is not unattended by danger. A considerable segment of the lower pole of the kidney may be solely dependent on this vessel for its circulation. An example occurred in a girl having recurrent attacks of right renal colic. Following division of an accessory lower polar vessel for intermittent hydronephrosis, there was a slight persistent serous discharge from the wound and some irregular elevation of temperature. After five weeks, a necrotic cast of about one-fourth of the kidney, entirely detached from the remaining renal tissue, was removed from the wound. The sinus rapidly healed.

Before tying off the vessel, the circulation should be temporarily

obstructed for several minutes and the lower pole observed. Should marked cyanosis occur, simple nephropexy, or nephropexy combined with suture of the ureter in a position to avoid angulation, may be attempted. In advanced dilatation, division of the ureter and anastomosis with the pelvis at its lowest point, below the level of the aberrant vessel, should be attempted in

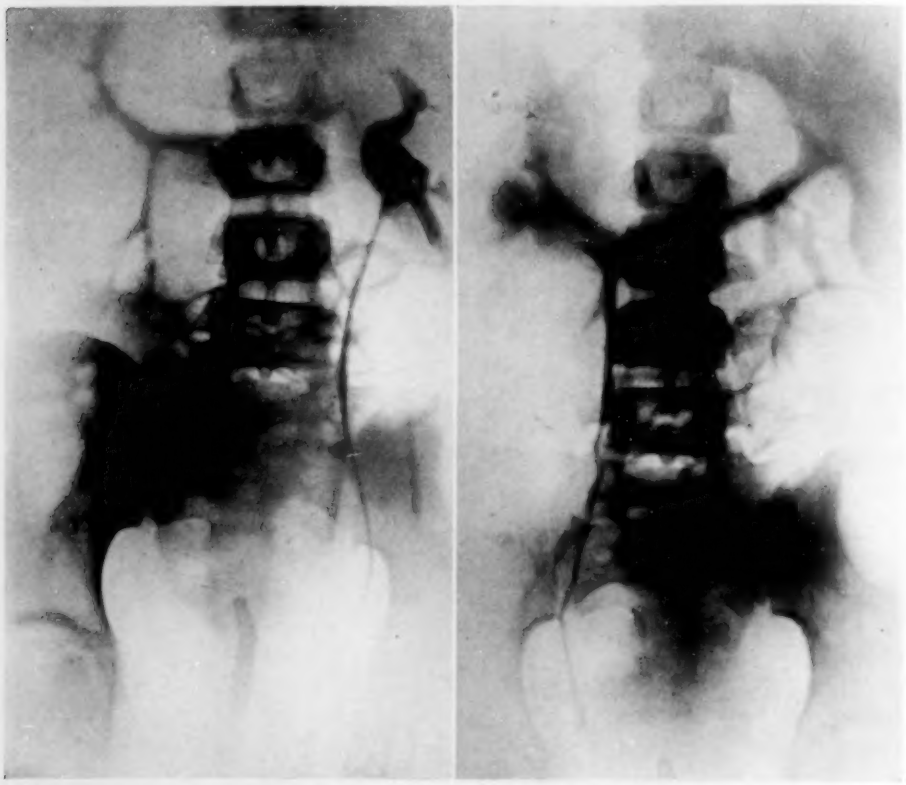


FIG. 10.—Accessory lower polar vessel. No. 36211. Female, ten years, April 26, 1926. Ureteropyelograms show marked left hydronephrosis and a normal ureter. There is a suggestion of slight hydronephrosis on the right side and the emptying time of the pelvis was delayed. Function of left kidney so greatly diminished it could not be estimated. Left nephrectomy revealed an accessory vein to the lower pole causing obstruction and consequent hydronephrosis and infection. Possibility of a similar anomaly on the right side.

preference to jeopardizing the viability of any considerable portion of the renal tissue.

Reduplication of the pelvis and ureter may be unilateral or bilateral and may be complete or incomplete. Where the duplication is complete the ureter from the upper pelvis empties by way of the lower of the two orifices in the bladder, while the ureter from the lower pelvis runs to the upper orifice. This necessitates a twining of the ureters about one another, and where they cross obstruction is apt to occur. Where duplication is incomplete, fusion of the two ureters may take place at any point between the lower pelvis and the bladder. Obstruction from the pressure of one ureter on the other is less likely to occur than in the complete variety, but either type is frequently accompanied by circulatory anomalies that may produce stasis.

Frequently duplication of the pelvis and ureter causes no stasis and gives rise to no symptoms. Attacks of pain from intermittent hydronephrosis or persistent pyuria from chronic pyelonephritis are the indications that call for urological investigation. The operative treatment demanded varies with the individual case. Freeing the ureter and ureteropexy may be sufficient to ensure proper drainage. Discission of some unimportant elements of the anomalous circulation, or section of one ureter and an end-to-side anastomosis of the proximal end into the other ureter may eliminate the stasis. Heminephrectomy might be attempted where the renal destruction is sharply limited to one-half the kidney. Finally, nephrectomy may be demanded by advanced hydro- or pyonephrosis.

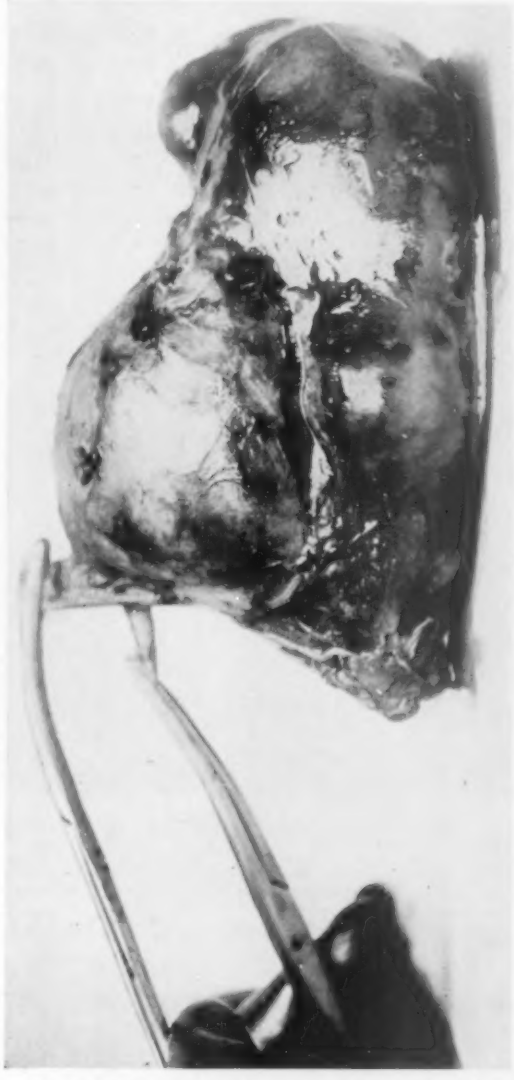


FIG. 11.—Operative specimen removed from case shown in Fig. 10. Hydronephrosis produced by accessory lower polar vessel.

Perhaps the most interesting condition met in childhood producing urinary stasis and destructive renal back-pressure is the enormous dilatation of the ureter called megalo-ureter, idiopathic dilatation and giant ureter. It is frequently bilateral. Although it might not properly be classed as an obstructive condition, as no stenosis of the ureteral orifice is present, it is evident that the aperture of the normal orifice is of insufficient calibre to adequately drain the tremendously distended ureter and pelvis present in these cases.

The etiology is still in doubt. It is most generally held to be a congenital anomaly, in many respects similar to megalo-colon. However in the latter condition the intestinal wall shows a great increase in the muscular layer. In the few specimens of megalo-ureter that we have had the opportunity to study microscopically, the thickening of the ureteral wall has been due to fibrosis

and inflammatory infiltration with very little increase in the muscular elements. Defective ennervation of the ureter diminishing the muscle tone and permitting dilatation, has been advanced as an explanatory theory. In the absence of any associated impairment of ennervation elsewhere it would seem improbable. No lesion of the lower urinary tract causing obstruction can be demonstrated and for this reason it is maintained that a dilatation secondary to a more distal obstruction cannot occur. Infection, for example

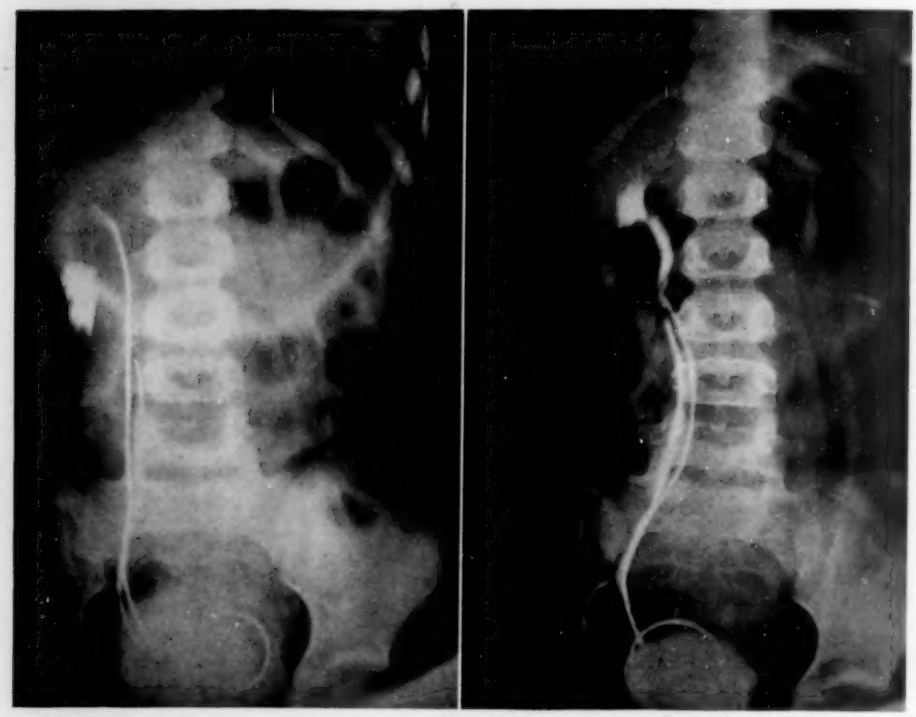


FIG. 12.—Reduplication of pelvis and ureter right. No. 77171. Female, six years, November 19, 1924. Uretero-pyelograms show slight dilatation of lower pelvis of the right kidney. Little blunting of calyces. Both ureters dilated from uretero-pelvic junction to point where they cross. Delay in emptying time. Obstruction caused by crossing of ureters, producing acute hydronephrosis. Attacks relieved by operation at which an anomalous renal circulation was demonstrated.

a long-standing pyelonephritis, has been advanced as the primary factor, the ureteral dilatation being a secondary manifestation. This view would not seem probable as many cases are seen where the urinary examinations over long periods of time show evidence of severe infection and yet the pyelo-ureterograms are essentially normal. On the other hand in occasional instances of megalo-ureter, the infection may appear to be relatively slight. A mechanical explanation is conceivable due to the obliquity of the course of the ureter through the bladder wall, causing angulation or valve action at the uretero-vesical junction. Ureteral stasis could induce hydro-ureter and a vicious cycle would be established, so that the greater the dilatation the more complete would be the obstruction.

Several cases of melago-ureter in boys have been observed where at first

sight no obstructive lesion was apparent. More careful study, however, revealed an obstruction in the posterior urethra. The findings in the bladder and upper urinary tract in these cases did not differ from those in which no such mechanical obstruction could be demonstrated. Even in the short female urethra valve-like folds of mucous membrane might occur that could obstruct and yet be very difficult of demonstration. In those cases where vesical retention does not occur, faulty implantation of the uretero-vesical junction could readily produce the ureteral dilatation. Mechanical obstruc-

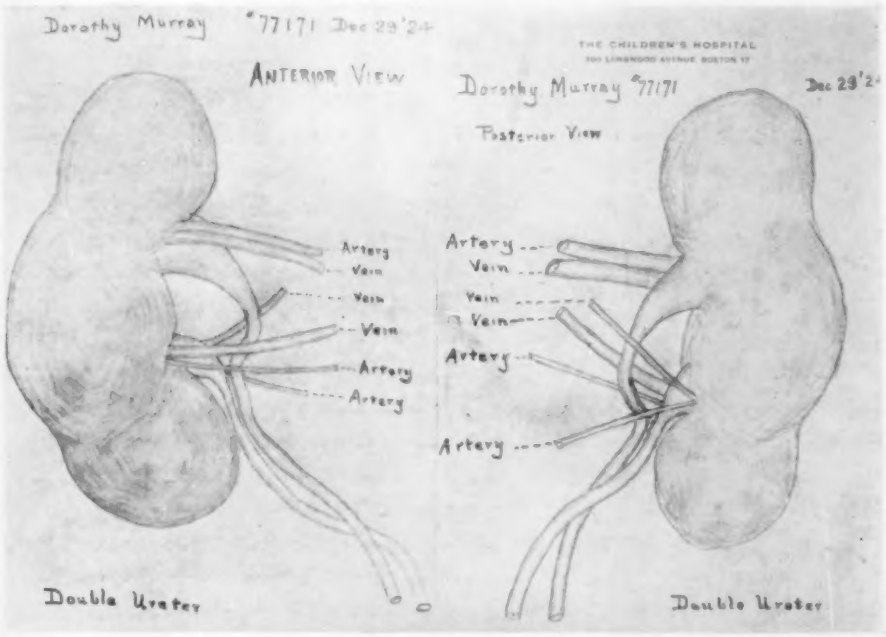


FIG. 13.—Schematic drawing of anomalous circulation found at operation in case of double ureter, shown in Fig. 12.

tion with a secondary infection would therefore appear adequate to explain cases of megalo-ureter.

With megalo-ureter the effects of long-continued absorption and toxæmia are evident. The children are underdeveloped and undernourished and may show mental retardation. Renal efficiency is markedly impaired and uræmic symptoms may develop. In one case multiple bilateral calculi were present. These were thought to be the result of infection and urinary stasis, and not the cause of the megalo-ureters.

Cystoscopy reveals a cystitis with injection of the mucous membrane and trabeculation similar to that seen in cases of excessive hydro-ureteronephrosis where a definite posterior urethral obstruction can be demonstrated. In the cases investigated, the ureter orifices have appeared normal and no inkling has been given of the condition present in the ureter. In the literature, however, a number of instances are reported in which the ureter orifices

were of the "golf hole" type, greatly dilated, the margins stiffened, and suggestive of the openings of diverticula. On insertion of the ureteral catheter a free regular flow of many cubic centimetres of urine is obtained. Ureterograms show enormously dilated and at times tortuous ureters, the dilatation extending from the bladder to the uretero-pelvic junction. At this point the calibre is usually somewhat narrowed. There is distention of the renal pelvis and at times this does not seem commensurate with the degree of hydro-ureter present. The calyces are mushroomed and there is advanced renal destruction.

The operative treatment of megalo-ureter has been unsatisfactory owing to the extensive renal damage previously sustained. A plastic on the ureteric orifice seems indicated, as the size of the normal opening is inadequate to drain the tremendous hydro-ureter above, which may attain the size of the large bowel. Unfortunately a cystostomy with slitting of the orifices, acccompanied by tube drainage of the kidneys through the ureters, is sooner or later followed in many instances by lighting up of the infection and anuric symptoms. Consequently bilateral nephrostomy must be considered in many of these cases before undertaking any operative measures on the ureters. Renal efficiency has been so far reduced by back pressure and infection that the margin of safety is very narrow. Infection with progressive renal impairment is practically impossible to check in the presence of a good urinary current through the dilated ureter and it may be found that a permanent double lumbar nephrostomy will be found to give the most comfort and the longest expectation of life. However the importance of a thorough and painstaking search for an obstructive lesion should be emphasized, as it seems probable from our experience that such a lesion may exist in a considerable number of the cases that have previously been classified as idiopathic dilatation of the ureter.

SUMMARY

Obstruction of the urinary outflow in childhood is far more common than has been generally recognized in the past.

Urinary stasis is followed sooner or later by infection and progressive renal destruction from back pressure and inflammatory changes.

Urinary obstructions are chiefly due to congenital anomalies and may be caused by intrinsic lesions of the urinary tract or by pressure from without.

Intrinsic lesions are situated most commonly at the uretero-pelvic junction, the vesical orifice or the region of the veramontanum in the male urethra.

Causes of pressure from without on the urinary tract are usually anomalous renal vessels or reduplication of the pelvis and ureter.

Megalo-ureter produces urinary stasis and renal impairment. The etiology is in doubt but it seems probable that an obscure obstructive lesion, either in the posterior urethra or at the uretero-vesical junction, associated with a

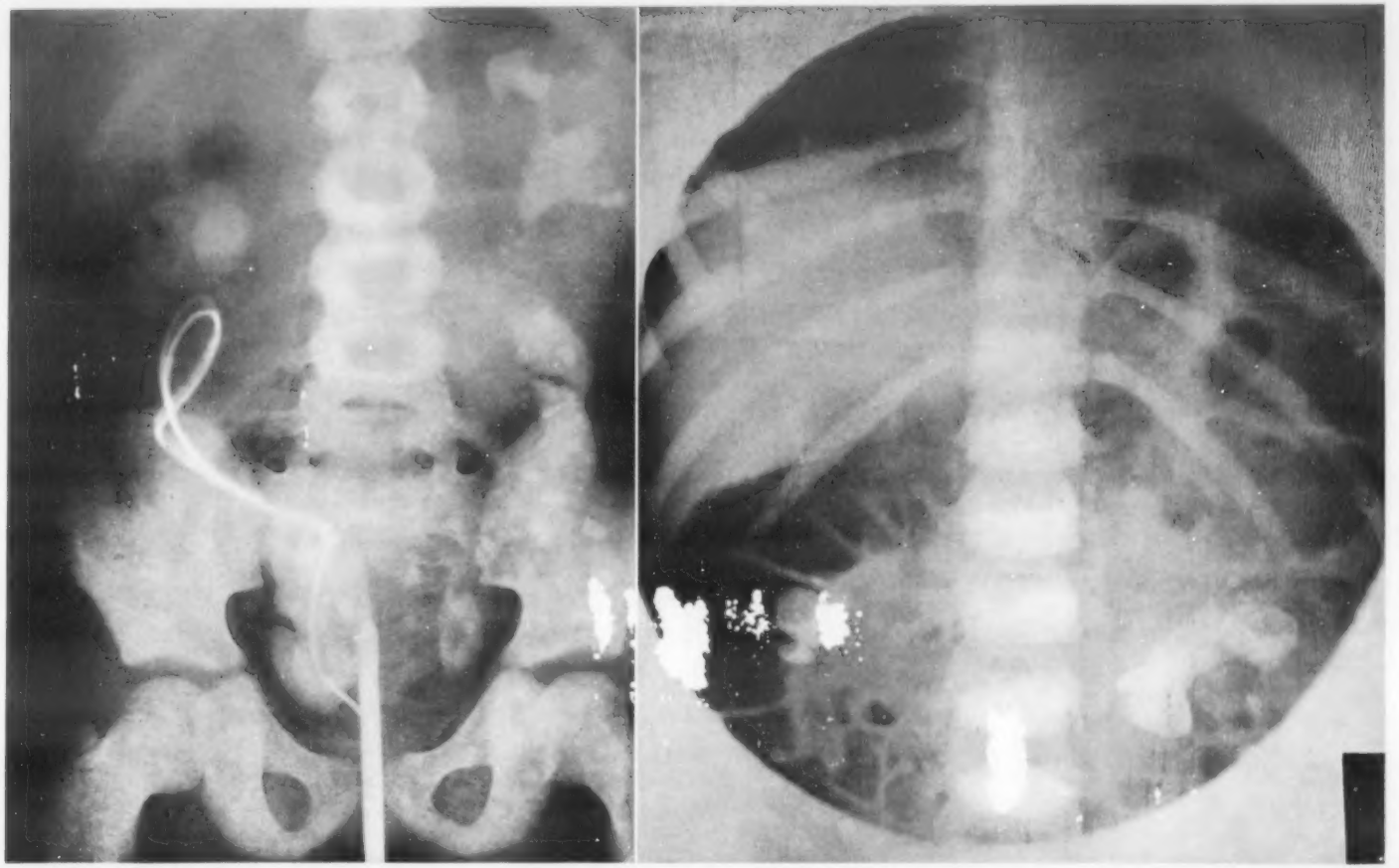


FIG. 14.—Megalo-ureter. Bilateral nephrolithiasis. No. 24222. Male, seven years, January 15, 1924. Uretero-pyelograms show enormous bilateral dilatation of the ureters and hydronephrosis. Calculi were present in the lower ends of both ureters and the pelves of both kidneys. Two years after the last operation for drainage of pyonephrosis and removal of multiple calculi, the patient died of uræmia. No mechanical obstruction was demonstrated at autopsy, but unfortunately the urethra was sectioned below the bladder without any careful investigation of the posterior urethra.

secondary infection, offers sufficient explanation. It is generally bilateral. The ureteral orifices are normal but are not competent to drain adequately the enormously dilated ureters.

In any type of lesion encountered it is imperative to relieve the obstruction to the urinary outflow at an early stage to prevent extensive renal destruction.

The destructive process is already far advanced in the majority of the cases before an attempt is made to establish an accurate diagnosis.

Cystoscopy, cystograms and uretero-pyelograms are indicated in childhood in all cases suggesting obstructive urinary lesions or showing persistent or recurrent pyuria.

ESSENTIAL THROMBOCYTOPENIC PURPURA—PURPURA HEMORRHAGICA AND ITS TREATMENT BY SPLENECTOMY *

By Edwin Beer, M.D.
of New York, N. Y.

During the last ten years our knowledge of the purpuras and the proper treatment of one type of purpura by splenectomy has advanced very rapidly. This particular type which is benefited by splenectomy was described as a clinical entity by Werlhof in the eighteenth century. Advance in the differentiation of the different types of purpura has been so slow that only fifteen years ago as prominent a clinician as Litten totally misunderstood the condition, and in an article in Modern Clinical Medicine [1] made the statement, "I believe strongly and absolutely that the individual purpuric diseases are not essentially different, but are due to the same cause and only vary in degree; that is, the varieties depend upon the intensity of the affection." This startling statement was made even though a superficial knowledge of the literature might have indicated that careful blood examinations made years before had already indicated that there were distinct differences between the types of purpura that presented in the clinic.

A Belgian histologist, Denys, in 1887, had already called attention to the fact that there was a low platelet count in some of these hemorrhagic diseases and a few years later, in 1890, Georges Hayem was able to confirm this interesting observation and at the same time called attention to the fact that although the blood coagulated, the clot did not contract. In the latter's important work on Diseases of the Blood,[2] published in 1900, these various facts are brought together and their significance emphasized. Whether Denys was the first to call attention to this low platelet count or whether this observation had been made by Brohm in 1883 and published in the dissertation of E. Kraus, as noted by Minkowski,[3] I have been unable to confirm. Be that, nevertheless as it may, Hayem deserves the credit for his intensive study of these purpuras, both the essential and the secondary, and his careful work seems to be the foundation stone of our present conception of the disease and of its recognition. He emphasized the following five peculiarities in the type of disease known as Werlhof's disease or essential purpura hemorrhagica:

First.—That there was no anatomical change which was appreciable in the red blood cells.

Second.—That there was a considerable diminution of the number of blood platelets and that those that were present were often of a large size.

Third.—There was no constant modification in the leucocytes. In only

* Presented before the Surgical Section of the New York Academy of Medicine, February, 1926.

one case was there an increase in these elements, quite independent of any blood disease.

Fourth.—That the blood coagulates normally but that the fibrinous reticulum remained either invisible or developed as fibrils of unusual size.

Fifth.—That there was an absence of retraction of the clot and secondary expression of the serum.

The pathognomonic and constant characteristic of this disease he saw in the diminution of the number of blood platelets and the absence of contractility of the clot with the usual expression of serum. He also called attention to the fact that there were numerous cases of purpura not associated with these two striking characteristics. A very complete classification of the different types of purpura, those with low platelet count, and those without, has been published only recently in the article by Doctors Brill and Rosenthal,[4] which reclassification confirms many of the excellent observations made twenty-five years ago by G. Hayem.

In 1912 Duke[5] called attention to the fact that the bleeding time may be greatly prolonged while the clotting time is normal. This prolongation of bleeding time after a pin prick may in some cases exceed an hour, whereas normally, bleeding ceases within three minutes.

Following in the lines of Hayem's original work, E. Frank,[6] in Germany, in 1915, practically rediscovered the work of the earlier Belgian and French students and called attention to the various conditions associated with purpura, hæmophilia, scurvy, various blood diseases, etc., and again emphasized the importance of the type associated with low platelet count, to which he gave the name of essential "thrombopenie."

In 1916, Kaznelson[7] had an opportunity to study several of these cases in which the patients had an enlarged spleen, and under the impression that the low platelet count might be due to a destructive action of the spleen upon the blood, he advocated its removal. Experimental work had already shown that the corpuscular elements of the blood, the platelets and red blood cells following splenectomy, normally are increased, in fact Alfred Hess, in a paper on the Consideration of the Reduction of Blood Platelets in Purpura, published February, 1917, in the proceedings of the Society for Experimental Biology and Medicine, quite independent of the publication of Kaznelson in Vienna, apparently arrived at the same conclusion. He states that it has been established that the removal of the spleen, both in men and animals, brings about a definite increase in the number of blood platelets. It would, therefore, "seem worthy of trial to perform a splenectomy immediately preceded by blood transfusion in severe cases of purpura where extreme therapeutic measures and repeated transfusions have been resorted to in vain." I have been told by Dr. E. Peterson, of this city, that in this year, 1917, in one or two cases of this type of purpura, Doctor Hess had referred the patients to him for splenectomy on the basis of the above conclusions. Fortunately, it was known from the literature that in cases of purpura hemorrhagic Hungarian surgeons had operated for appendicular infection without encoun-

tering any difficulty, so that it was evident that though dealing with patients who were liable to bleeding, often uncontrollable bleeding, the operative interference of splenectomy would not be contra-indicated because of inability to control oozing from the incised wound. The first case of Kaznelson was published in the latter part of 1916, in the Wiener klinische Wochenschrift,[7] the operation being a splenectomy done by Professor Schloffer in Prague.

The patient presented the clinical picture of extreme thrombocytopenic purpura. She was a female of thirty-six and had been under observation for many years for chronic recurring hemorrhages. She had severe epistaxis petechiæ in the skin, ecchymoses, and had since youth the bleeding tendency. Ten years before the operation she had had severe bleeding from her genitalia, from her nose, from her gums, and general petechiæ. Her hæmoglobin had been as low as 10 per cent., and there was a sudden crisis with improvement, but the epistaxis and petechiæ frequently developed. In 1910, she had severe bleeding after parturition and thereafter had repeated attacks of severe menorrhagia. In 1913, the tendency to bleeding still persisted, and in 1916, the year of admission and operation, there was an uncontrollable epistaxis which dominated the picture. On physical examination her spleen was three fingers' breadth below the ribs, her blood pressure was practically normal, no lymphatic enlargement, no tenderness of the sternum or tibia. Her blood examination showed red blood cells 3,792,000, white blood cells 6710, and the platelets, which were almost exclusively giant forms, numbered 200. Coagulation began in three minutes but there was no clot reaction even at forty-eight hours. The patient's nose was packed for six weeks before the bleeding could be controlled. Petechiæ developed all the time under observation and there was bleeding from the gums. After removal of the spleen, which was a comparatively simple procedure, the change in the clinical picture was most astounding. The bleeding tendency stopped. The platelets rose to 500,000, the bleeding time was shortened, the patient prior to the splenectomy used to bleed from the slightest needle prick, whereas now there was difficulty in getting a specimen on pricking the finger. Moreover, the clot reacted normally. The patient was reported, four weeks after operation, as showing a marked improvement, if not a cure, by splenectomy, of essential purpura hemorrhagica or Werlhof's disease.

Since this startling report a great many cases, well over fifty, some perhaps of doubtful validity and not definitely proven cases of the disease under discussion, have been published in the literature of Austria, Germany and America, and a few isolated cases in England, the Dominions and France.

The disease under discussion seems to run at least two very different courses. The type that is usually encountered is the chronic recurrent or relapsing type, and it is in this type that splenectomy seems to be particularly useful, even though at the present time it is not absolutely certain that it is all that has been credited to it, namely that it leads regularly to a permanent cure. The other type, the acute type, is a much more rapidly progressing disease and judging from the published reports, splenectomy in these cases is of very questionable value. Even if its field be limited in this way, it may be a definitely life-saving measure in the chronic cases as the repeated hemorrhages and recurring attacks in the chronic cases may, if untreated by splenectomy, eventually lead to the death of the patient.

As far as the clinical picture of this disease is concerned, it differs altogether from hæmophilia in that it is not hereditary. It seems to occur more frequently in the young and hemorrhages may occur in almost any part of

the body, as small petechiæ or ecchymotic spots in and under the skin, as bleeding from the gums, stomach, intestines, from the genitalia, from the urinary organs, and from the nose and throat. The laboratory findings in the cases that have been studied all seem to show what Hayem originally called attention to, namely a low platelet count, an absence of retraction of the clot, a normal coagulation time usually associated with a prolonged bleeding time which may be instead of the average three to five minutes as long as one hour or more. In true hæmophilia the platelets are not diminished, the tourniquet produces no petechiæ, coagulation time is prolonged and bleeding time is usually normal.[8]

A variety of theories as to the origin of thrombocytopenia and its relation to the uncontrollable bleeding have been advanced. It has been suggested that the disease is primarily in the marrow and that there is a defective formation of platelets. Others have suggested that toxins in the circulation or otherwise destroy the platelets that are normally present in normal amounts in the blood. Kaznelson, in view of the fact that in his cases the spleen was enlarged, thought perhaps there was a lytic process which destroyed the platelets in the spleen. None of these theories has been entirely satisfactory, and Minkowski called attention to the fact that in his case the spleen was small and normal, pathologically. A platelet count of the blood aspirated from the splenic artery and from the splenic vein just prior to removal of the spleen in one of the patients operated upon by me, did not show any such change in the number of platelets as the theory of Kaznelson would suggest, Doctor Rosenthal having found practically the same number per cubic millimetre in both blood specimens. (Case III.) Further study along these lines is indicated. Apparently in only one other case has a note been made in the report of a comparative study of the two bloods, the splenic vein and the splenic artery blood, without any convincing difference in the number of platelets. Another interesting feature of this disease is that after the splenectomy, although there is a preliminary rise in the number of platelets in the blood, very frequently the platelet count drops again to very near the low number that had been present prior to operation and still the patient is rarely troubled with any severe bleeding. In one of the five cases reported in this paper it was noticed that whereas before the operation, adrenalin locally applied had but very little effect upon the capillary oozing, after splenectomy the oozing from the granulations of the drainage tract was quickly controlled by the application of adrenalin. This isolated observation might suggest that while the spleen is in, normal contractility of the capillaries is defective, and in view of the fact that the disturbance in the number and perhaps quality of the platelets has a distinct bearing upon blood coagulation, the combination of the disturbance in the capillaries plus the disturbance in the coagulability of the blood due to the thrombocytopenia may underly the pathogenesis of these varied bleeding phenomena. The peculiar swing in the platelet count to high figures after splenectomy and down again to low figures, Minkowski has suggested might be due to the influence of the remaining reticulo endothelial system or to

accessory spleens which possibly produce a lytic substance which destroys the platelets much as the original spleen had done, according to the viewpoint of Kaznelson.

Herewith I submit reports of five typical cases, four of chronic relapsing thrombocytopenic purpura in which the end results as seen months to years after the operation are most gratifying, the patients having been restored to complete health, as well as one acute case in which splenectomy was done but in which an early fatality ensued. The records of these cases I owe to the coöperation of Dr. N. Rosenthal.

Case I, reported by Brill and Rosenthal, *Archives of Internal Medicine*, 1923, p. 946:

Case I.—A boy, aged fifteen, was admitted November 22, 1922, complaining of bleeding from the nose and vomiting of blood. The present illness began in April, 1919, when the patient had an attack of tonsillitis followed by hemorrhages into the skin, bleeding from the gums, painful joints, vomiting and pain in the upper part of the abdomen. He also had irritability of the eyes, weakness, fever, chills and sweats. He remained in the hospital for one month, after which he was apparently well except for an occasional ecchymosis following some slight trauma, until May, 1922, when he received a blow on the nose. This was followed by a severe epistaxis which continued for several hours. The bleeding was stopped by means of a tampon saturated with fresh normal blood. The skin hemorrhages had become more frequent since. In July, 1922, while drinking milk, blood began to issue from the anterior and posterior nares and soon the patient vomited blood and food. Some hemorrhagic areas again appeared on the skin. Röntgen-ray therapy was applied to the splenic region, with apparently good results. His condition improved. He lived a quiet life until November 22, 1922, when he was again struck on the nose, and he had been bleeding and vomiting blood ever since.

Physical Examination.—The patient was a well-developed and fairly well-nourished boy with marked pallor. There were a few petechiæ in the conjunctivæ of both lower lids. The teeth were in fair condition. The gums were spongy and bleeding, the tonsils large and covered with hemorrhagic spots. The heart was not enlarged. There was a systolic thrill and murmur at the apex. The spleen was not palpable, but it was large to percussion. There were numerous petechiæ over the back, chest, abdomen, thighs and legs.

On November 26, 1922, the blood count was: hæmoglobin, 45 per cent.; red cells, 2,584,000; white cells, 10,000; platelets, 10,000 (plasma); polymorphonuclear neutrophils, 71.6 per cent.; polymorphonuclear eosinophils, 1.6 per cent.; polymorphonuclear basophils, 0.3 per cent.; lymphocytes, 15.3 per cent.; and monocytes, 11 per cent. The coagulation time of the blood was ten minutes; the bleeding time four and one-half minutes. The tourniquet test was slightly positive. There was no clot retraction. The patient had secondary anæmia, thrombocytopenia and monocytosis. The blood picture was characteristic of essential thrombocytopenia.

During the following month there were a succession of hemorrhages from nares and gums, producing an anæmia so marked that transfusion was done December 17, 500 c.c. being injected. There were no hemorrhages after this transfusion, but successive crops of petechiæ formed. December 23, the hæmoglobin content was 28 per cent.; red cells, 2,010,000; platelets, 24,000. December 29, a second transfusion was done, 450 c.c. being injected. The following day, December 30, 1922, splenectomy was performed by Dr. E. Beer through a subcostal incision. Tube drainage of subphrenic space. There was profuse oozing of the wound. Continuous oozing from the nose occurred during the anæsthesia.

A soft slightly enlarged spleen with omental adhesions between the stomach and

hilus was found. The adhesions were doubly divided and cut. The spleen was delivered with some difficulty. The hilus was ligated and cut, taking special care not to include the adherent stomach.

Summary of pathologic report by Dr. F. S. Mandlebaum: The macroscopic specimen consisted of a moderately enlarged spleen weighing 300 gm. and measuring 14 x 7.5 x 3 cm. It was elastic and cut easily. Malpighian bodies were visible. Microscopic examination showed only hypertrophy. No blood platelets were found.

Immediately after the removal of the spleen, all oozing of blood stopped. The bleeding before splenectomy was profuse at the end of six minutes when it was stopped. The bleeding time during manipulation at the hilus was six minutes; immediately after splenectomy, three minutes; two hours after splenectomy, three minutes; eight hours after splenectomy, three minutes; and fifteen hours after splenectomy, two and one-half minutes.

January 9: Some oozing from granulations about drainage tract which stopped at once with application of adrenalin.

January 19, 1923: The patient was out of bed. Many petechiæ appeared on the legs and a few on the right lower conjunctivæ.

February 3, 1923: A few petechiæ on the face and lower legs appeared from time to time. The gums had improved; there was no sponginess and no bleeding.

February 8, 1923: There were hypostatic petechiæ on the legs only. The general condition was excellent. The hæmoglobin content was 76 per cent.

February 14, 1923: For the first time clot retraction was present. There was a thrombocytopenia and slight positive capillary resistance test. The petechiæ were disappearing from the legs. There had been no hemorrhages since February 9, 1923.

February 17, 1923: The patient was discharged well.

Blood changes following splenectomy:

(1) Hæmoglobin and red blood cells: The transfusion of 500 c.c. before splenectomy raised the hæmoglobin to 48 per cent. and the transfusion given immediately after the operation produced a further rise to 60 per cent. and a rise to 3,232,000 red blood cells. This gradually dropped during the first four days to 38 per cent. hæmoglobin and 2,832,000 red blood cells. Improvement then began and at the last examination (April 6, 1923) the hæmoglobin was 81 per cent. and the red blood cells were 4,840,000. Normoblasts and Howell-Jolly red cells were occasionally present.

(2) White blood cells: Just before the operation there was a leucocytosis of 22,000; six hours after the operation the leucocytes were 36,000, and on the following day they rose to 55,000. The differential blood picture after the post-operative polynucleosis showed a persistent monocytosis (increase of the large mononuclear and transitionals).

(3) Blood platelets: The day following the operation there was a slight rise to 31,200; then a gradual fall to 1000 on the third day after the operation. After this there was a gradual increase to 10,000 and then to about 20,000. The morphology remained about the same. The day following the operation a few giant blood platelets appeared in the smears.

(4) Bleeding time: For a month and a half this was prolonged, usually over two minutes, and even as long as twelve minutes. This became normal (two to three minutes).

(5) Tourniquet test (capillary resistance): This was constantly positive until the third month after the operation. It then became constantly negative.

(6) Clot retraction: There was no clot retraction for six weeks after the operation. This appeared on February 14, 1923, and slight clot retraction remained present, although the blood platelets remained low. It is interesting to note that the blood of this patient never showed clot retraction on previous examinations.

Summary.—This was a case of chronic thrombocytopenia of four years' duration. The patient's condition became worse as time went on; the bleeding was more frequent and more severe. Splenectomy brought about a turn for the better and the patient has steadily improved since.

The patient was again seen in April, 1923. He had no hemorrhages since he left the hospital. He had gained weight and strength steadily. Static purpura of the legs did not occur. Examination of the blood still showed a thrombocytopenia (blood platelets, 22,000), but all other evidence of the previous condition was absent. The capillary resistance test was negative and clot retraction was present. February, 1925, presented at the New York Surgical Society, the patient is entirely well. February, 1926 patient in excellent condition three years after splenectomy.

CASE II.—E. G.: A young girl, aged seventeen years, was admitted on October 9, 1924, to the First Medical Service, complaining of uterine bleeding for nine months, black and blue spots of skin and bleeding from mouth two weeks, and bloody urine two days. About nine months before admission to the hospital, she noticed that her menses occurred three days before the usual date and that the period lasted several days longer with profuse bleeding. At that time she began to find black and blue spots on her skin, especially after the slightest bruise. About March, 1924, she began to bleed from the gums. October 7 she felt some pain in her left loin and since then she noticed that her urine was bloody. The blood in the urine has become less.

She appeared a well-nourished and well-developed girl, not acutely ill. Petechial hemorrhages were present on the mucous membranes of the mouth (gums, lips and fauces) and petechiæ and ecchymoses were present on all parts of the skin. The heart and lungs showed no abnormalities. The liver was not felt, but the spleen was easily felt, and extended two fingers below the costal margin.

Laboratory Examinations. (1) Urine—bloody at first, but later clear. (2) Blood, Wassermann—negative.

Blood

Hæmoglobin	94%	Bleeding time	42 min.
Red blood-cells	5,120,000	Coagulation time	8 min.
White blood-cells	12,600	Tourniquet test	Positive
Platelets	10,000	Clot retraction	None
Polys. neut.	64.6%	Temperature	98° to 99°
Lymphocytes	31.3%	Pulse	88/120
Monocytes	4.0%	Respiration	20–24

Platelets very large.

The condition of the patient did not improve on the usual medical treatment and after a week in the hospital she began to menstruate profusely. She complained of feeling weak and this was reflected in the blood examinations. The hæmoglobin and red blood cells began to drop rapidly. October 18, 1924, the hæmoglobin was 69 per cent. and the red blood cells were 3,890,000. Pallor was becoming marked and the hemorrhages in the skin and mucous membranes increased. Splenectomy was done October 24, 1924, by Dr. Edwin Beer before the members of the Clinical Congress of Surgeons. Ether was used as an anæsthetic. A long left subcostal incision was made. The spleen was found high up under the diaphragm, adherent posteriorly and anteriorly. The spleen was not much enlarged and did not extend below the ribs. At the hilum of the spleen an accessory spleen, the size of a cherry, was found. No intraperitoneal bleeding was noticed. The patient stood the operation well. The pathologist reported no abnormal changes in the spleen except a relative increase in the number of Malpighian bodies.

The post-operative course was very stormy. The hæmoglobin kept steadily going down and November 1 reached 33 per cent. A blood transfusion of 500 c.c. was again given, but with little effect. The progressive fall in the hæmoglobin was due to the menorrhagia which was not checked by the splenectomy. The bleeding into the skin and mucous membranes, however, stopped. November 8, radiotherapy, to the hypophysial region to check the hemorrhage from the uterus was done. After this her bleeding became less and a week later the uterine bleeding stopped. She was then transferred to

the medical side November 16, with the wound almost healed. From then on she improved steadily and was discharged well December 3, 1924. Her hæmoglobin on discharge was 62 per cent.

A few of the blood examinations done since the splenectomy show the following:

	Oct. 29, 1924	Nov. 5, 1924	Dec. 11, 1924
Hæmoglobin	35%	29%	62%
Red cells	2,464,000	1,960,000	4,320,000
White cells	30,400	50,800	18,000
Platelets	12,000	10,000	80,000
Polys. neut.	85.3%	90%	78.5%
Lymphocytes	12.6%	8%	11.0%
Monocytes	2.0%	2%	8.0%
Coagulation time	8 min.		9 min.
Bleeding time	6 min.	7 min.	2 min.
Tourniquet test	Neg.	Neg.	Neg.
Clot retraction	None (24 hrs.)		Normal

The blood platelets do not show any great increase at first, compared to their number before operation. The leucocytosis has persisted. The blood picture is gradually assuming a normal aspect. Presented at the New York Surgical Society, ANNALS OF SURGERY, June, 1925.

The platelet count went up after operation and then dropped to pre-operative figures, but never returned to normal.

Patient continues in excellent health—no more bleeding.

CASE III.—A man, M. S., eighteen years of age, was admitted June 6, 1925, with the history summarized as follows:

Summary.—1. Measles in childhood. 2. Eczema in infancy—duration one year. 3. Influenza eight years ago. 4. Frequent sore throats up to three years ago. 5. Tonsillectomy three years ago. 6. Papular eruption associated with pruritis and bleeding one year ago. Duration two months. Bled easily and freely. Spontaneous disappearance. 7. Onset of present illness two and one-half months ago. 8. Onset with small hemorrhagic spots on feet, legs, forearms and chest. No pain or other sensations. 9. Eruption shows regression under treatment but recurred. 10. Two months ago began to spit blood. Unassociated with pain in chest, fever, chills or cough or sweat. Lasted one day. 11. Epistaxis two weeks ago. 12. Hæmaturia thirteen days ago. 13. Bleeding from gums at present. 14. Pain in left knee since yesterday. 15. Splenectomy advised at Bellevue.

Present Illness.—Two and a half months ago, found small hemorrhagic pin point to pin head sized flat spots on the feet. No pain, tingling or other sensations. Came out on the legs at the same time. Later in the day noted same condition on the forearms. The next day it came out on the chest. Felt perfectly well during this time. At the end of a week went to M.D. who gave patient a tonic and advised rest. Took tonic and rested for one week. Improvement noted during that week. Whole skin had previously appeared red and hemorrhagic at the site of the eruption but at end of week the skin showed a very great improvement so that comparatively few hemorrhagic spots were left. Returned to work. (Feeder on a printing machine; comes in contact with the metal of the linotype machine and the printed sheets.) Second day after returning to work, noticed he was spitting blood. No cough. No pain in chest. Thought blood came from throat. Blood was bright red. Eruption on body returned. Returned to M.D. Not relieved. Went to Bellevue three weeks ago. Was there fifteen days. Was given capsules. Eruption disappeared under medication but returned three days later. Two weeks ago nose began to bleed. Next day urine became bloody. Lasted two days. Never had blood in stool. Left Bellevue six days ago. Returned for observation few days ago. Told he needed a splenectomy. Bleeding from nose has ceased. Bleeding from mouth (gums) continues.

Hemorrhagic eruption persists. Since yesterday has pain in left knee. Lost six and one-half pounds in three weeks.

The patient is poorly nourished. General examination negative. The skin presents generalized multiple petechiæ, most numerous over chest. Hæmoglobin index, 65 per cent.

Bleeding time, eighteen and one-half minutes. Clotting time, six and one-half minutes. Tourniquet test, negative. Clot retraction, none after twenty-two hours, none after three days.

Red cells, 3,200,000; white cells, 9,875; polymorphonuclears, 65 per cent.; lymphocytes, 23 per cent.; eosinophiles, 2 per cent.; large monocytes, 10 per cent.

Blood-pressure, 110/60; hæmoglobin, 70 per cent.; platelets, 96,000. First sound at third interspace left of sternum reduplicated.

June 9, 1925

Hæmoglobin	76%	Polys. neut.	71%	Myelocytes	8%
Red cells	3,840,000	Polys. eos.	6%	Coagulation time ..	7 min.
White cells	20,600	Polys. bas.	2%	Bleeding time	14 min.
Platelets	5,000	Lymphocytes	13%	Tourniquet test ..	Sl. Pos.
				Clot retraction	None

The blood picture is typical for thrombocytopenic purpura hemorrhagica. The leucocytosis is probably the result of hemorrhage. Splenectomy is indicated provided that tuberculosis or any cause for symptomatic purpura are ruled out. Vide post. X-ray report on lungs which were negative.

The operation was done July 16, 1925, through a curved incision from close to ensiform process down to rectus sheath; across flat muscles of abdominal wall laterally between tip of twelfth rib and anterior superior spine.

The spleen with four accessory spleens at hilum was removed without any particular difficulty, vasa brevia were first doubly tied and splenic vein and artery exposed. After freeing tail of pancreas, on surface of which a small vein was tied, pedicle of spleen was ligated and dropped back. There was no oozing and abdominal wall was closed with modified "figure of 8."

August 4, 1925, Blood Examination

Hæmoglobin	55%	Baso.	4%
Red cells	3,610,000	Myelocytes	4%
White cells	13,000	Myeloblasts	2%
Polys.	71%	Bleeding time	2½ min.
Lymphocytes	12%	Coagulation time	2½ min.
Monos.	3%	Tourniquet time	Neg.
Eosino.	4%	Platelets	130,000

Wound healing slightly disturbed by serous accumulation at lower angle requiring period of drainage. Rapid recovery, sutures removed on the tenth day, examination of the spleen after removal showed normal structure. Pulp shows myeloid metaplasia. February, 1926 the man is in perfect health.

Case IV.—M. A., a boy thirteen years of age was admitted September 11, 1925, suffering from epistaxis. Bleeding from gums, ecchymoses and purpuric spots on skin, four weeks.

No history of bleeding tendencies in family so far as patient knows. No history of other chronic familial disease. Father and mother living and well, one sister living and well.

Had mumps at age of eight. Had slight epistaxis previously only on rare occasions—perhaps once in two to three years and then of very short duration. No previous bleeding from the gums, no previous purpuric manifestations. No injuries or operations. Systems entirely negative. Head, eyes, ears, nose and throat negative. Cardiorespiratory—no

dyspnœa or palpitations, no cough. G. I.—appetite always good. Bowels regular. No digestive disorder. No melena or hæmatemesis. No dysuria—no hæmaturia.

Four weeks ago, the boy began to bleed from the nose. For a full week the bleeding persisted as a steady ooze and then stopped. Three weeks ago, for the first time he began to notice large and small ecchymotic blotches appearing over his body, chiefly in the lower extremities and hip regions. As these disappeared in the course of several days, new ones appeared. At the same time, crops of small pinhead sized purpuric spots began to make their appearance, most profusely over the extremities especially the lower but also over the rest of his skin. During these three weeks, he has also had bleeding from the gums. Has had no hæmoptysis, or hæmatemesis, has noted no blood in his stool or urine, has had no pains in the abdomen nor elsewhere. Since the onset of his illness, he has complained of slight dizziness at times when suddenly changing position. No visual disturbances. Thinks he has lost weight but does not feel particularly weaker than formerly. For two days, he has noted dryness and roughness of skin of hands and toes with vesiculation and desquamation of the superficial layers of the skin. No itching. Appetite is good, bowels are regular. Has had no digestive disorders. No urinary symptoms. No fever or chills. General examination negative.

He is a well-developed and well-nourished boy of thirteen, with no marked pallor, cyanosis or jaundice. Distributed over the body, posteriorly on legs, shoulders and back innumerable small cutaneous hemorrhages, most about 1 to 1½ mm. wide. In about ten places on the body, back, legs and arms are fading ecchymotic areas in stages of resorption, most about 2 to 3 cm. wide.

August 12, 1925, Blood Examination

Hæmoglobin ..	67%	Polys. neut.	64%	Coagulation time—5 min.
Red cells	3,472,000	Polys. bas.	2%	Bleeding time—greater than 10 min. (stopped)
White cells ...	10,500	Lymphocytes ...	31%	Tourniquet test—positive (2 min.)
Platelets	20,000	Monocytes	3%	Retraction of clot very slow at end of 18 hours

Impression.—Typical blood picture of purpura hemorrhagica.

Pre-operative diagnosis.—Thrombocytopenic purpura.

Operation.—Splenectomy by Doctor Beer. Sub-costal incision. Stomach pulled mesially exposing the vasæ breviæ which were carefully tied and the vessels of the pedicle were in turn tied with chromic, pushing the tail of the pancreas from the hilus. The opening in the peritoneum, over the tail of the pancreas, was drawn together after the removal of the spleen. There was no particular bleeding from anywhere except the parietes which was controlled by ligatures before opening the peritoneum and by two layers of chromic sutures in closing the parietes. The deeper layer through muscle and peritoneum being continuous interlocking and the second layer being interrupted fascio-muscular. The skin was closed with silk.

Primary wound—healing.

August 21, 1925, Blood Examination

Hæmoglobin	75%	Polymorphonuclears	74%
Red cells	3,850,000	Lymphocytes	12%
White cells	26,550	Monocytes	14%
Platelets	550,000		

September 6, 1925—Final Note: Typical case of thrombocytopenic purpura hemorrhagica treated by splenectomy. Discharged to P. P. Well.

Urine examinations (7) negative.

February, 1926, patient is in excellent health.

CASE V.—A woman, twenty-two years of age, was admitted November 27, 1924 complaining of bleeding from nose on and off for four months and also increasing weakness. Past history—Negative. Six months ago, she started feeling weak, lost her appetite and began to have severe pains in front part of head with buzzing in the ears. She became gradually worse until four months ago when she had a severe nose bleed after blowing her nose; this lasted for two hours. She bled again one month later and again three weeks ago. She became weaker after each attack of nose bleeding. For past three weeks has been vomiting and noted the presence of blood in the vomitus one day before admission. Two weeks, the stools were tarry. No red spots were noticed in the skin. There was no bleeding from any other region except the nose. Her menstruation which stopped a week ago began again on day of admission.

She was a well-developed, pale woman with hemorrhagic blebs on lips. Conjunctivæ very pale. Fundi show multiple hemorrhages. The gums were swollen and bleeding actively. Numerous hemorrhages of mucous membrane of mouth and tongue. The tonsils were covered with numerous small hemorrhagic spots. The neck showed no glandular enlargements. Lungs were negative. The heart was normal in size; systolic murmurs at apex and base were not transmitted. Liver was soft, felt three fingers below costal margin. Spleen was firm, rounded edge felt three fingers below costal margin. Skin showed numerous petechial hemorrhages over neck, back, chest, abdomen and extremities. Symmetrical vitiliginous lesions over the back, chest, neck, breasts and lower extremities.

The admission diagnosis rested between acute purpura hemorrhagica and acute leucopenic myeloid leukemia.

Clinical Course.—Temperature varied between 99 and 103 before splenectomy. Pulse—80–140. Respiration—24–28.

November 27, 1924—Blood Examination on Admission (Doctor Rosenthal)

Hæmoglobin	22%	Lymphocytes	23%
Red cells	1,168,000	Monocytes	11%
White cells	7,400	Normoblasts—3 per 100 white cells	
Platelets	2,500	Coagulation time—4 min.	
Polys. neut.	62%	Bleeding time—10 min. (stopped on account	
Polys. bas.	11%	of profuse bleeding)	
Myelocytes n.	2%	Capillary resistance test—Positive	
Myeloblasts	1%	Clot retraction—None (48 hours)	

The blood picture is suggestive of leucopenic myeloid leukemia and symptomatic purpura hemorrhagica.

November 28, 1924—A direct transfusion of 800 c.c. was given. The hæmoglobin rose to 33 per cent. The general condition remained poor. No fresh petechiæ but gums were still swollen and bleeding.

December 1, as her condition remained the same and as the outlook was not encouraging, a splenectomy was decided upon to stop the hemorrhagic tendency although the blood picture suggested the presence of leukemia on account of the rapidly progressive anemia and the presence of a few premature myeloid granulocytes, the opinion was held that the condition may also be an essential thrombocytopenic purpura hemorrhagica. As no other form of therapy was available, splenectomy was considered as a last resort. A preliminary transfusion of 800 c.c. was given prior to the operation to put patient in condition to permit the operative procedure. The splenectomy was done by Dr. E. Beer, December 2, 1924, through a subcostal incision, ten inches long.

Procedure.—The enlarged spleen gradually delivered as vessels were tied. The gastro-splenic ligament was very short and had to be ligated to the greater curvature of the stomach. The splenic artery and vein were easily ligated. The capsule of the tail of the pancreas was intimately adherent to the hilum and had to be stripped away

leaving an oozing area—this was buried with a few sutures. This oozing could not be completely controlled. Drainage—Gauze covered with rubber dam.

A blood transfusion of 700 c.c. was given just before the operation and another transfusion of 750 c.c. was given immediately after the operation as the patient was in shock.

Three hours after the operation the patient died. Permission for autopsy could not be obtained.

REFERENCES

[1] Modern Clinical Medicine. D. Appleton and Company, 1911.
[2] Hayem, G.: Maladies du Sang, 1900.
[3] Minkowski, O.: Mediz. Klinik, December 7, 1919.
[4] Brill, N. E., and Rosenthal, N.: American Journal of the Medical Sciences. Oct., 1923.
[5] Duke: Archiv. Int. Medicine, 1912.
[6] Frank, E.: Berl. Klin. Wochenschrift, 1915.
[7] Kaznelson, P.: Wiener Klin. Wochenschrift, 1916, No. 46.
[8] Hess, A. F.: Archives Int. Medicine, 1916–1917, p. 203.

ACUTE PANCREATITIS

By Irvin Abell, M.D.
of Louisville, Ky.

In an experience embracing more than 900 operations on the biliary tract the writer has encountered ten cases of acute pancreatitis. All but one occurred in the course of more or less prolonged cholecystitis, presenting varying degrees of gall-bladder pathology. The term "acute pancreatitis" as here employed, covers at least three distinct conditions, acute pancreatic necrosis, acute hemorrhagic pancreatitis and pancreatic abscess. It is believed that the latter is but an advanced stage of the first two, the patient surviving the acute onset with subsequent infection and suppuration of damaged pancreatic tissue and hemorrhagic deposit resulting in abscess formation. Of the ten cases, five occurred in men and five in women: five were markedly obese, one moderately so, three were of muscular development and one quite emaciated. The average age of the patients was 39.7 years, the individual ages being 17, 32, 34, 39, 50, 51, 51, 54, 56 and 64.

Previous History.—Four of the ten patients had had typhoid fever and all but one gave a definite history of gall-bladder disease. Duration of symptoms referable to gall-bladder: two cases 1 year; two cases 2 years; one case 3 years; one case 6 years; one case 8 years; one case 10 years; and one case 20 years; average 5 years, 10 months. Seven gave a history of gall-bladder colics while in the remaining three the colic that occurred with the onset of the pancreatitis was the initial one. All had suffered digestive disturbances for varying periods of time. One had undergone an operation for removal of stones and drainage of gall-bladder, six years before coming under my care, at which time he presented a second crop of calculi in his gall-bladder with an acute hemorrhagic pancreatitis.

Symptoms Referable to Acute Involvement of Pancreas: Duration.—Two, two days; three, three days; one, four days; two, five days; one, seven days, and one, ten days. In seven the symptoms attracting attention to the pancreas developed at periods varying from one to four weeks following a gall-bladder colic, there being a subsidence of pain and other subjective symptoms referable to the gall-bladder before onset of those referable to pancreas or else a continuation of gall-bladder symptoms with those of pancreas gaining in intensity and consequent ascendancy. In three the pain accompanying the onset of the pancreatitis was the initial one, the previous symptoms being of reflex digestive character of mild degree.

Pain.—The pain in seven was acute, severe and agonizing, accompanied by incessant nausea and vomiting, representing the ultra-acute type; in three it was much less severe, the cases pursuing a milder course. In five it was referred to the epigastrium and right subcostal region, being described as similar to that experienced with previous attacks of gall-bladder colic; in two it was felt in the right subcostal region, extending across the upper abdomen

to the left subcostal area; in three the maximum intensity was noted in the left subcostal area. Cyanosis was observed in but two. The pulse in three was under 100, 88, 90 and 96; in seven over 100, 110, 115, two, 120, 138, and two 140. Temperature varied from 99.5 to 102. Systolic blood-pressure from 100 to 154. Considering the pathology found at operation the leucocyte counts were not high, 5300, 6800, 9800, 11,800, 12,000, 12,700, 13,300, 14,800, 15,300, 18,800. Such blood findings are in harmony with the belief that the extensive destruction of pancreatic tissue is due to an activation of the trypsinogen within the pancreas rather than to actual bacterial attack.

Urine.—The urine in all cases showed the presence of albumin; none showed the presence of sugar. Bile was present in five, casts in four, microscopic blood in seven and microscopic pus in nine.

Mass.—In five patients no mass was detected, while in the remaining five an enlargement was distinctly palpable: in three the mass was felt in the right hypochondrium, in one in the left hypochondrium and in one it extended transversely across the abdomen.

Pre-operative Diagnosis.—The diagnosis in five was acute cholecystitis; in one of these the detection of a mass at the site of the pancreas after the patient was anæsthetized led to a correct diagnosis before the incision was made. In one the condition was thought to be acute intestinal obstruction, in three a correct diagnosis of acute pancreatitis was made, while in one no diagnosis other than acute abdomen was made.

Morbid Anatomy.—Free fluid was found in the greater peritoneal cavity in three cases, in one of which it was bile-tinged and of large quantity: hemorrhagic fluid exudate was present in the lesser peritoneal cavity in seven instances varying in amount from a few to 2600 c.c.

Rather widely disseminated areas of fat necrosis were encountered in four and none detected in six. The gall-bladders were visibly diseased in all ten cases and nine contained calculi: the common ducts of all upon palpation were negative for stones.

In three the pancreatic lesion presented as a hemorrhagic pancreatitis, in two the hemorrhagic infiltration being confined to the pancreas and tissues immediately adjacent; in one of moderate size, in the other of such extent as to form an oblong mass across the abdomen which was palpable through a rather thick wall; in the third the infiltration involved not only the pancreas, but extended into the subhepatic and right perirenal spaces and into and behind the ascending mesocolon as far as the cæcum. In one case the pancreas was enlarged to approximately four times the normal size, nodular and elastic, bile-tinged fluid in lesser and greater cavities, thickening and œdema of gastrohepatic omentum and pancreatic fatty capsule, all of which bled on manipulation, although no visible hemorrhagic deposit was present.

Two presented a moderate increase in size of head and right half of body with marked enlargement of left half of body and tail, the mass involving the gastric surface of spleen, the lesser peritoneal cavity and the transverse mesocolon: no visible hemorrhagic deposit present.

In two the head of the pancreas was markedly enlarged and in one at operation was thought to be a subacute pancreatitis accompanying an acute cholecystitis: the patient died nine days after operation and autopsy revealed acute pancreatic necrosis involving the head and part of the body of pancreas.

In one, the lesion presented as a retroperitoneal pancreatic abscess holding six ounces of pus in which were flocculi, caseous masses and bits of sloughing pancreatic tissue. Cultures from pus showed colon bacillus.

In one, the condition presented as a pseudocyst of the lesser peritoneal cavity, 2600 c.c. of hemorrhagic fluid, sterile upon culture, being removed: the layer of peritoneum forming the posterior wall of the lesser cavity immediately over the pancreas had disappeared, the partly necrotic pancreas being exposed to view upon removal of fluid.

Operations.—In eight cases a drainage of the pancreas was employed, the route of approach being through the gastrocolic omentum in four and the gastrohepatic omentum in four: in six of the eight, cholecystostomy with removal of calculi was done, in one cholecystectomy was done, in one the condition of patient was so precarious that the gall-bladder pathology was not disturbed. Of the two in which pancreatostomy was not employed, in one the pancreatic inflammation involved the left half of the body and the tail of the pancreas without apparent necrosis and a removal of calculi and cholecystostomy was relied upon to give drainage: in the other, the pancreatic pathology was thought to be a subacute inflammation complicating cholecystitis: a cholecystectomy with common duct drainage was done followed by death, autopsy showing acute pancreatic necrosis.

SYNOPSIS OF CASE HISTORIES

Case I.—No. 5237—February 23, 1917. Male, age thirty-two. History gall-bladder colics. Duration present illness three days. Pain, nausea, vomiting, tenderness right upper quadrant; jaundice present; bile, albumin, casts and pus in urine. White cell count 5300, polymorphonuclears 64.7, small lymphocytes 23.3, large lymphocytes 11.5. Tentative diagnosis: Cholecystitis. Operation: head of pancreas shows marked enlargement; removal large distended gall-bladder containing calculi; drainage common duct through cystic duct. Post-operative history: no bile from drainage tube; bleeding from bowel, stomach, gums and drainage tube; severe epigastric pain, collapse and death on ninth day after operation. Autopsy: Acute pancreatic necrosis. Pathological report: Chronic cholecystitis, acute and chronic pancreatitis.

Case II.—No. 10,097—March 16, 1921. Male, age fifty-one. Operation for gall-stones six years ago. History of colics before and since operation. Last colic seven days ago, since when has been confined to bed. Pain in epigastrium radiating to gall-bladder area and to right renal area. Temperature 98.2, pulse, 90. Slight jaundice. Rigidity and tenderness right upper quadrant, most marked tenderness in right renal area. Urine contains bile, albumin, casts, microscopic blood and pus. White cell count 6800. Tentative diagnosis: cholecystitis with recurrent calculi. Operation: adherent mass consisting of gall-bladder, duodenum, pylorus, colon and omentum. Upon separation hemorrhagic infiltration with areas of necrosis in pancreas, subhepatic and right renal spaces, and ascending mesocolon as far as cæcum, gall-bladder containing calculi removed and common duct drained through cystic duct. Gastrohepatic omentum opened and pancreas

drained with gauze cigarette; cigarette drains placed in subhepatic and right renal spaces. Recovery.

Case III.—No. 13,119—November 8, 1922. Female, age thirty-nine. History digestive disturbance; no colics. Acute onset forty-eight hours ago; pain, nausea, vomiting, constipation, slight cyanosis. Pulse 120, temperature 102. Leucocytes 15,300. Polymorphonuclears 86.5 per cent. Abdomen shows slight mass in left upper quadrant. Urine shows albumin, casts, microscopic blood and pus. Tentative diagnosis; intestinal obstruction. Operation: Acute hemorrhagic pancreatitis; pancreatostomy through gastrohepatic omentum; cholecystostomy with removal of stones. Recovery.

Case IV.—No. 14,099—October 15, 1923. Male, age fifty-two. History of gall-bladder colics and reflex digestive disturbance. Has had recurrent attacks of iridocyclitis for years. Last colic four weeks ago. Since onset of last or present illness has had continual pain in right upper quadrant. For past week has had fever, 101 to 102°. Tender mass in right upper quadrant. Leucocyte count 12,800, polymorphonuclears 78 per cent. Urine shows albumin and microscopic pus. Tentative diagnosis: Cholecystitis. Operation: Gall-bladder contains stones and is not adherent to mass. Cholecystostomy with removal of calculi. Mass corresponds to pancreas and overlying omentum shows multiple areas of fat necrosis. Mass approached through gastrocolic omentum and is found projecting into lesser peritoneal cavity. Opened and evacuated of six ounces of pus showing colon bacillus on culture; necrotic putty-like masses of pancreatic tissue removed from abscess cavity; drainage. Recovery.

Case V.—No. 15,678—March 31, 1924. Female, age thirty-four. Colics and digestive disturbance for more than one year, marked and associated with vomiting at intervals for past year. At times vomitus has contained blood. Has been bedfast for past six months. Weight one year ago 173, present 100. In October, 1923, first noted swelling or mass in upper abdomen which at times has disappeared; has been constantly present for past month. Pules 138, temperature 100. Fluctuating mass occupying upper abdomen between the costal margins, extending from ensiform to point below umbilicus, most marked to left of midline. Blood shows hæmoglobin 68, red blood-cells 3,170,000, leucocytes 13,300. Urine shows albumin, microscopic blood and pus. Tentative diagnosis: Cholecystitis, pancreatitis with pseudocyst of lesser peritoneal cavity. Operation: Local anæsthesia; lesser peritoneal cavity opened above stomach and evacuated of 2600 c.c. of hemorrhagic fluid sterile on culture. Pancreas shows necrosis of surface exposed in sac. Gall-bladder contains multiple calculi and is not disturbed. Edges of incision in lesser cavity are sewn to parietal peritoneum and lesser cavity drained with tubes. Recovery.

Case VI.—No. 16,360—September 1, 1924. Female, age fifty. Digestive discomfort, epigastric pain and colics for three years. For past six weeks has noted increase in pain which has been practically continuous with evening temperature of 100 to 101. Abdomen has increased in size. While in hospital for further study was seized with acute pain, nausea, vomiting and fever rose to 102; pulse to 140. Abdomen shows the presence of fluid, is tender and rigid over gall-bladder, extending to left of midline. Leucocyte count on entering hospital 11,800, after onset of acute attack 18,100; polymorphonuclears 82.5 per cent. Urine shows albumin, microscopic pus and blood. Tentative diagnosis: Cholecystitis, acute pancreatitis. Operation: Bile-tinged free fluid in greater cavity. Gall-bladder is thick-walled, œdematous and contains stones. Cholecystostomy with removal of stones. Pancreas is greatly enlarged, nodular and soft in consistence; adjacent tissue is œdematous, hyperæmic and bleeds on slightest manipulation. Gastrocolic omentum opened and drains placed down to head of pancreas. Recovery.

Case VII.—No. 16,713—November 3, 1924. Male, age sixty-four. History of gall-bladder colics and digestive disturbance over a period of twenty years. Mild colics three weeks ago. Present acute illness began with severe colic six days ago; pain has necessitated opiates continually since. Nausea and vomiting marked. Abdomen exquisitely tender in epigastrium and under right costal margin. Pulse 108, temperature 100. Leucocytes 9800. Urine shows albumin, microscopic pus and blood. Tentative diagnosis:

Cholecystitis. When under the anæsthetic mass could be felt extending across abdomen corresponding to site of pancreas; added diagnosis of acute pancreatitis made. Operation: General peritoneal cavity contains free clear fluid. Lesser peritoneal cavity opened through gastrocolic omentum contains hemorrhagic fluid, multiple areas of fat necrosis in omentum and mesocolon. Pancreas is imbedded in hemorrhagic exudate and presents multiple areas of necrosis. Cholecystostomy with removal of calculi; pancreatostomy with tampon drainage. Recovery. During convalescence this patient had several hemorrhages from drainage tract requiring packing for control.

Case VIII.—No. 17,157—December 18, 1925. Female, age fifty-one. History of gall-bladder colics and digestive disturbance over a period of years. Duration of present illness four days; severe colic, nausea, vomiting, slight jaundice; greatest intensity of pain noted in left upper quadrant. In previous attacks or colics pain had always been noted in right upper quadrant. Pulse 96, temperature 101; tender over entire epigastrium, most marked to left midline. Leucocytes 12,700. Urine shows albumin, bile, microscopic blood and pus. Tentative diagnosis: cholecystitis. Operation: gall-bladder thick-walled, non-adherent, contains multiple stones. Pancreas is enlarged, the left half of body and the tail are greatly enlarged and imbedded in inflammatory infiltration which involves the hilum of spleen and transverse mesocolon. No hemorrhagic deposit present. Cholecystostomy with removal of stones. Recovery.

Case IX.—No. 17,796—June 17, 1925. Female, age seventeen. History of digestive upset of one week's duration one year ago. Similar disturbance for past three weeks characterized by burning, fulness and discomfort in epigastrium. Twenty-eight hours before admittance to hospital suffered severe, acute pain in left upper abdomen radiating to axilla and back, accompanied with marked vomiting. Temperature 101, pulse 120, slight jaundice, stony rigidity over entire epigastrium, tenderness most marked to left of midline. Bile, albumin and pus cells in urine. White cell count 14,800, polymorphonuclears 80, small lymphocytes 18, large lymphocytes 2. Tentative diagnosis: Acute abdomen. Operation: general cavity contains free fluid. Gall-bladder shows subacute inflammation with adherent omentum and colon. Mass at site of left half of pancreas approximately 4 x 3 x 2 inches is exposed through gastrohepatic omentum; mass consists of nodular enlargement of part of body and tail of pancreas with adhesion of and inflammatory infiltration into transverse mesocolon. Pancreatostomy with drainage through gastrohepatic omentum, Cholecystostomy. Recovery.

Case X.—No. 18,893—December 5, 1925. Male, age fifty-six. History of gall-bladder colics and digestive disturbance over a period of years. Present illness began with acute onset forty hours ago—pain, nausea, vomiting, collapse: temperature 101, pulse 140; leucocytes 12,000, polmorphonuclears 81, small lymphocytes 11, large lymphocytes 7, eosinophiles 1. Urine shows albumin, casts, microscopic pus and blood. Tender mass under right costal margin. Tentative diagnosis: acute gangrenous cholecystitis. Operation: acute gangrenous cholecystitis with multiple calculi present. Many areas of fat necrosis in omentum and transverse mesocolon. Mass at site of head of pancreas which is exposed through gastro-hepatic omentum; turbid fluid in lesser cavity. Pancreas presents multiple areas of necrosis. Drainage through gastrohepatic omentum. Cholecystostomy. Death on eighth day following operation.

SUMMARY

Acute pancreatic necrosis, acute hemorrhagic pancreatitis and pancreatic abscess are not separate clinical entities, but represent different stages of the same process, the origin of which is not entirely clear. The rapid destruction of pancreatic tissue is due to the activation of trypsinogen within the gland itself; normally this is done by the enterokinase in the duodenum. The most logical explanation for its activation within the pancreas is that it is due to a

retrograde injection of infected bile or duodenal contents through the ducts of Wirsung and Santorini as well as by the minute hemorrhages and bacterial toxins resulting from a pancreatic lymphangitis. Biliary tract infections have been present in more than fifty per cent. of the reported cases, in one hundred per cent. of the series herewith reported. The lymphatics draining the gall-bladder and bile ducts are in intimate association with the lymphatics of the head of the pancreas before they join the aortic group. Infection following this path readily enters the head of the pancreas where resultant inflammation and minute hemorrhages may readily activate the pancreatic ferment. The powerful digestant action of the ferment upon the blood-vessels of the pancreas doubtless explains the presence of marked hemorrhagic deposit while the absorption of the autolyzed pancreas, toxic proteoses is in large measure responsible for the shock and early toxic manifestations.

The areas of fat necrosis commonly seen in the peritoneum, root of mesentery, mesocolon and omentum are due to the action of ferments in the escaped pancreatic secretion upon the fat molecule, breaking it up into its component glycerine and fatty acids. Cases reported in which such areas have been observed in the pericardial and extrapleural fat would indicate that these ferments are capable of transportation by lymph or blood stream.

There are no pathognomonic symptoms; pain, vomiting and collapse being the most important encountered. The physical signs will depend on the stage of the disease at which the patient is seen; in some cases the lack of symptoms and physical signs is remarkable when compared with the extent and severity of the local lesion.

Laboratory examinations are of but little aid in reaching a diagnosis; for this reliance must be had upon the history of previous upper abdominal disease, the present symptoms and physical findings. Pain radiating from the right costal margin across the upper abdomen, tenderness following the course of the pancreas, pain and tenderness to left of midline and the detection of a mass in the pancreatic area are beacon lights when elicited. After all it is not so important to make a correct diagnosis of acute pancreatitis as it is to make a correct diagnosis of an acute surgical lesion in the upper abdomen: the predominance of symptoms at and above the umbilicus will usually permit of this localization when prompt operation will direct one to the pathology. The earlier the operation the less the destruction of the pancreas, the less the absorption of toxic proteoses the less the peritonitis and consequently the greater the number of recoveries. The indications are to relieve tension, to stop hemorrhage, to prevent leakage and to afford drainage: the fact that the pancreas has no proper capsule, being imbedded in loose retroperitoneal cellular tissue and fat permits of rapid extension of inflammatory infiltration: pancreatostomy with application of tampon and tube drains in and around the focus of pancreatic destruction will best fulfill these indications. The drainage of the gall-bladder, when the condition of the patient permits, is a worthwhile procedure in promoting recovery and securing immunity from further attacks.

CYSTS OF THE OMENTUM*

By William John Ryan, M.D.

of Philadelphia, Pa.

from the department of surgery, st. mary's hospital, philadelphia

Since 1852, when Gairdner [1] reported an autopsy specimen before the Pathological Society of London there have been but 44 cases of omental cysts reported in the literature. It would seem, therefore, that this condition is a rare one and warranted an analysis of the cases reported as well as a detailed report of a case coming under our care.

In 1908, Hasbrouck [2] reported a case of supposed cyst of the omentum, but on analyzing his description and that of the pathologist it is apparent that it was not a cyst but an endothelioma. His review of the literature disclosed the fact that only 19 cases had been reported previous to the one he described. His case, like ours, had its origin in the omentum between the stomach and the transverse colon.

In 1911, Dowd [3] presented the report of a case in the Annals of Surgery and assisted by Doctor Farr tabulated the cases reported previous to this, with Hasbrouck's paper as a basis. They were able to collect 37 cases, 24 were in females and 13 in males, 16 patients 20 years or over and 24 in children.

The symptoms were variable and none were marked except a prominence in the abdomen, and in some, embarrassed respiration from pressure. Only two followed direct trauma to the abdominal wall, Gooding's case, one year after trauma and Cotman's three months after receiving a blow. The cases were all very incompletely reported and there was lack of complete description of the cyst wall and fluid. Hence it was impossible to reason accurately as to their origin. He calls attention to the large number giving evidence of blood in their contents; four had coagulated blood and eighteen contained fluid so dark in color that they apparently had contained blood.

His own case followed straining at stool. Operation disclosed a cyst attached to pelvic peritoneum below. A pedicle above proved to be twisted omentum. It contained a fluid, pale in color, of a specific gravity of 1008; albumin 1 per cent. by bulk, and a few red blood-cells which apparently got in through the operation. He thinks the cyst originally was a hæmatoma, in which the hæmoglobin was absorbed and the blood-cells degenerated.

Stillman,[4] in 1911 reported a case of his own and one described by Reginal Fitz in the Lane Lecture in San Francisco. Both of these, however, were in women who had fibroid tumors of the uterus to which the omentum was adherent. He states that only 19 of the reported cases could be classed as true cystoma.

---

* Read before the Philadelphia Academy of Surgery, May 3, 1926.

Frank [5] thinks these tumors should be called cystomata because they have secreting cyst spaces and a definite neoplastic formation. He reviewed the previous literature and analyzed Stillman's paper and a report of three cases by Markoe and McPherson.[6] In the latter he rightfully eliminated case one, because it was a hæmatoma in the omentum which had become twisted between the round ligament and a fibrous band remaining from a previous suspension operation.

As to the origin, it would certainly seem from a study of our case that they are lymphatic. Jacobi, quoted in Frank's paper, believes they are lymphatic in origin and are either the result of dilatation of lymph-veins or a cystic degeneration of lymph-nodes.

Outerbridge [7] sums up his opinion as follows: While the possibility of the origin of some cases of true cysts of the omentum from embryonal rests or from the surface peritoneum cannot be denied positively, it may be considered demonstrated beyond doubt that in other instances the lymph-vessels are the starting point for such growths, and this latter explanation would appear to be the one which would apply to the majority of cases. Dr. W. T. Reese, the pathologist who examined our case, thinks they are of lymphatic origin. The question may be raised here as to how the thin-walled cysts in our case came to have blood in them. We feel certain they contained clear fluid, but, as they increased in size the blood-vessels were thinned out and finally ruptured, allowing blood to flow into the cyst cavity.

Of the 19 cases selected by Stillman as being true cystomata of the omentum only two were of the gastro-colic omentum. We are privileged to add one to this number.

No. 3401.—Name George S., age four. Referred by Dr. John G. Sabol. Admitted May 3, 1925 on account of swelling of abdomen which embarrassed respiration.

The parents of the child state that since birth they thought its abdomen was rather large and consulted a physician who told them it was just fat. About two years before admission the abdomen was tapped and a large amount of brownish fluid was drawn off with great relief for the boy and marked reduction in the size of the abdomen. At this time percussion disclosed a tympanitic note in both flanks, and dullness in mid-abdomen. No mass could be palpated. The fluid slowly recurred and a year later the abdomen was tapped again to relieve discomfort. Operation was refused at this time. Three weeks before admission a mass could be detected in epigastrium but was not very clearly felt because of fullness of the abdomen.

There has been no vomiting at any time. Constipation could be relieved by purgative. Appetite good. Except for the respiratory embarrassment the child was perfectly well. Loss of weight but slight and that within the last four months. Except for the history of being delivered with forceps there was no record of any trauma. Had diphtheria twice. Parents living and well.

Physical Examination.—A male child, sitting up in bed, well developed and fairly well nourished. Respiration somewhat labored. Except for palpable anterior and posterior cervical glands the neck was negative.

Chest.—The lower chest was much wider than normal, so that the angle between the costal cartilages and sternum was an obtuse one. Expansion was limited. *Lungs.*—

normal. *Heart.*—regular and of good volume, no murmurs present. *Abdomen.*—markedly enlarged and gives a dull tone to percussion all over. In the upper mid-abdomen a distinct mass is palpable. It feels smooth on its surface and can be moved around a little. It has a doughy feel. Posteriorly and at the level of the lower ribs is the only place a Tympanitic note can be found. Peristalsis could not be heard. Liver dullness could not be made out. X-ray threw no light on diagnosis. Day after admission, abdomen tapped again with hope that with most of the fluid out a more definite examination could be made of the mass detected. As the fluid was drawn the mass became more distinct and was moved down in the abdomen until it was at the level of the umbilicus. It was round, smooth, about four to five inches in diameter and appeared to be attached behind by a pedicle. A tentative diagnosis of sarcoma of the omentum was made. The fluid drawn off was dark brown, almost like coffee ground vomitus.

Operation.—Abdomen opened through midline incision. The mass felt at examination presented itself as a grayish-white cyst with a thick wall. A small needle withdrew clear, straw-colored fluid. Surrounding this cyst were two large and a number of small cysts. They were dark in color and very thin-walled, slightly transparent. No fluid could be found in the cavity. These cysts filled the entire abdomen and on investigation they were traced to the gastro-colic omentum. The white, thick-walled one was attached to the greater curvature of the stomach for about an inch. The intestines were packed posteriorly and upward. The greater omentum was small. Pedicle was clamped and the multiple cysts removed. The abdomen was closed by through and through because of poor condition. Three hundred c.c. of 3 per cent. glucose were given intravenously and 20 mm. of adrenalin. Recovery uneventful.

Pathological Report.—Specimen consists of a group of cysts, joined together, three of which are whitish in color and thick-walled. Six of them are slate-colored and had thin walls, attached to them is the great omentum which lies free among the cysts.

The largest cyst is one of the thin-walled, slate-colored ones. It is kidney-shaped, its wall is 1 mm. in thickness. In its wall and in the other thin-walled cysts many small, rounded, nodular masses can be felt, which project into the cyst cavity and seem to follow the blood-vessels and lymphatics. They vary in size from one to five mm. in diameter. They are very numerous. The fluid in the slate-colored cysts was dark brown in color, contained a large amount of globulin and albumin, was negative for sugar and positive for blood with the Benzidin test. Microscopically this fluid contained a large number of red blood-cells.

The thick-walled cysts were whitish in color and were very fibrous to touch. The walls varied from two to four mm. in thickness. The fluid was clear and straw-colored. There was only a faint trace of albumin and no sugar. An occasional red blood-cell could be found. Some of the round, fibrous bodies could be felt in these thick-walled cysts but were only few in number. In some areas they became linear elevations which were very dense and fibrous.

Microscopically.—Thick-walled cysts. The inner lining shows a single layer of endothelial cells which are supported by fibrous, connective tissue which shows a large amount of œdema separating the strands. There is a chronic inflammatory process in this portion of the cyst wall. Here are numerous small blood-vessels and lymphatics; infiltration with small lymphocytes and an occasional polymorphonuclear leucocyte. Below this area in the cyst wall there is a dense fibrosis which completes the cyst wall and which is covered by a single, very thin layer of flattened endothelium.

Thin-walled Cysts.—Œdema is found only in spots and is much less than in the thick-walled cysts. There is very much less evidence of an inflammatory process. Some of the blood-vessels show on the inner wall and are no doubt the source of the blood in the cyst cavity. The rounded bodies are thickened, fibrotic areas in the meshes of which

are blood-vessels and lymphatics which have been obliterated or which show dense fibrosis of the wall.

Diagnosis.—Large multilocular cysts of omentum, probably of lymphatic origin.

REFERENCES

[1] Gairdner: Trans. Path. Soc., London, 1852, vol. iii, p. 374.
[2] Hasbrouck: Annals of Surgery, 1908, vol. xlvii, p. 206.
[3] Dowd: Annals of Surgery, 1911, vol. liv, p. 617.
[4] Stillman, S.: J. A. M. A., 1911, vol. lvii, p. 726.
[5] Frank: Am. Jour. Obstetrics, 1913, vol. lxvii, p. 501.
[6] Markoe and McPherson: Bull. Lying-In Hosp., N. Y., 1912, vol. viii, p. 70.
[7] Outerbridge: Trans. Phila. Acad. Surg., 1915.
[8] Norris, C. C.: The Omentum. Its Anat., Hist. and Phys. U. of Pa. Med. Bull., 1908.
[9] Cotman: British Med. Jour., 1902, vol. i, p. 1267.
[10] Funk: Surg., Gyn. and Obstet., 1914, vol. xvii, p. 70.
[11] Jacoby: Trans., Assn. Am. Phys., 1901, vol. xvi, p. 232.
[12] Rodman: Internat. Clinics, 1909, Annals of Surgery, 1909, vol. xlix, p. 437.
[13] Bell: Long Island Med. Jour., 1920, vol. xiv, p. 397.

INTESTINAL OBSTRUCTION*

By John B. Deaver, M.D.
of Philadelphia, Pa.

The steady advance in medical and surgical knowledge and technic has seen a progressive decline in the mortality of many diseases admitting of surgical treatment, but the death-rate in acute intestinal obstruction, all cases considered, remains practically stationary, at the appalling and incriminating figure of about fifty per cent. Although it is difficult to reach an exact figure, as the results in small series vary greatly and there are few large series of reliable statistics available, the ideal mortality in this condition should not exceed ten per cent. Where the responsibility lies for such an excessive and unnecessary death-rate is a problem worthy of our best study and analysis.

Acute intestinal obstruction is one of the most serious emergencies which the physician is called upon to diagnose and the surgeon asked to treat, and the deadliest factor in the condition lies in delay, whether it be delay in diagnosis or in the operative treatment. Only rarely does the surgeon have the opportunity of operating early upon a case of acute obstruction. As a rule, the surgical procedure in the early cases is simple and quickly performed and the mortality low. But with every passing hour of delay, the patient's toxicity is increased, his resistance is lowered and the surgeon's task thus is made more difficult. In the later stage, the resection demanded is often so extensive that the ebbing tide of life recedes forever under the added strain.

I have been prompted to discuss the subject of intestinal obstruction for a number of reasons. Among these are the present high death-rate; the comparative ease of early diagnosis and the advantages of early operation; the dangers of late pathology, which in the more common forms of acute abdominal ailments is responsible for the majority of cases of acute obstruction, and finally, the frequent error of confusing post-operative peritonitis and obstruction.

Why is the present death-rate fifty per cent. instead of ten per cent.? The answer is: early diagnosis has not been made. And why has an early diagnosis not been made? Because the naked abdomen has not been carefully scrutinized and carefully auscultated and palpated, including examination per rectum and per vaginam.

Far too many diagnoses of the acute abdomen are made through the clothes, and too often the pain-assuaging drug, morphia, is administered before the lesion responsible for the pain has been determined. Not to give morphia after the diagnosis has been made is inhuman, but to give it before the patient has been carefully examined from every angle is unpardonable.

Another reason for the high death-rate from operation for obstruction is

* Read before the Bronx County Medical Society, New York City, March 17, 1926.

the administration of purgatives. While this is reprehensible, attempting to empty the bowel by enema is justifiable, and if successful may rule out obstruction. But it should be borne in mind that a bowel movement obtained by enema means nothing unless the symptoms and signs have subsided and flatus is being passed and normal peristalsis has been restored.

The diagnosis must begin with a careful history followed by a most careful interpretation of the same. This cannot be stressed too strongly. Some years ago I had the privilege of being called in consultation with the late Dr. Reginald Fitz, of Boston, and the late Dr. John H. Musser, in a case of serious upper abdominal disease. Doctor Fitz spent one hour in interrogating the attending physician, the late Doctor Jurist, and making notes, and when he had finished going over the notes, he remarked, "That patient has an acute pancreatitis and I am now ready to see him." He spent only five minutes in examining the patient and then remarked: "Gentlemen, I am ready to confer with you." Upon returning to the consultation room he again said, "Your patient has an acute suppurative pancreatitis, and I advise operation." His advice was acted upon and the diagnosis confirmed at operation. The patient recovered, but later developed diabetes and died six years after operation.

An early diagnosis of obstruction, not the diagnosis of the variety of obstruction, should, in the majority of cases, be possible by correct interpretation of the history and careful examination.

In eliciting a history the questions asked should be: What is your occupation, having in mind lead colic. Have you ever had any sickness like this before? Were you prefectly well, or in your usual health, until the occurrence of the present trouble? Do you attribute this to anything in the way of indiscretion in diet, drinking, etc.? Have you, or have you ever had a hernia or rupture? Have you ever had an abdominal operation? Was the pain preceded by vomiting or a feeling of sick stomach? Is the pain steady or does it come and go? When did your bowels move last? Is the pain accompanied or followed by the passing of gas or the desire to have a bowel movement? Do you know whether the stools contained blood or mucus? Are you able to point to a spot where the pain is most intense? If the pain is paroxysmal, does taking a deep breath or coughing excite or aggravate it? Are you more comfortable when lying perfectly quiet or do you feel better when you change your position?

In examining the acute abdomen caused by obstruction, auscultation plays as important a rôle as palpation. It is here especially that the surgeon as well as the internist should possess the art of abdominal auscultation. Personally, I prefer the ear to the stethoscope, but the head must be applied gently, since its presence on the sore abdomen will make the patient more uncomfortable than if the stethoscope is used. The medical interne is recognized by the dangling rubber tubes of the stethoscope which he carries, usually in the hip pocket of his white trousers, and the surgical interne by the

stomach tube dangling from his coat pocket, both so useful in the diagnosis and treatment of abdominal surgical conditions.

The majority of operations for intestinal obstruction, exclusive of strangulated hernia, are caused by pathology the result of a previous abdominal operation or a previous peritoneal inflammation. Therefore obstruction should be thought of in the presence of acute abdominal symptoms if examination shows a scar of a previous operation. It should be superfluous to mention the appendix in this connection, but I find that the possibility of appendicitis as the cause of the symptoms is often overlooked by the attending physician and also by the surgeon.

The operation if made early should be short and curative because of the ease with which the pathology can be exposed and disposed of, thus avoiding a prolonged or complicated operation. What makes the surgeon's work easy produces less strain upon the patient's endurance, so that recovery is almost certain. The earlier the operation, before there is marked distention of the bowel proximal to the site of obstruction, the easier and quicker the site of obstruction can be found, which means less intraperitoneal traumatism, and less hesitancy as to what best to do.

The possible lesions in post-operative intestinal obstruction, that is, if occurring before convalescence has taken place, are: peritonitis, adhesions, secondary or residual abscess, most often following acute perforative or suppurative appendicitis. Recently the literature has contained reports of acute obstruction following posterior gastro-enterostomy where a knuckle of bowel has passed into the lesser peritoneal cavity between the wall of the stomach and the margin of the opening made in the transverse mesocolon for delivery of the portion of the stomach to be anastomosed. I have never had this occur, as I make it a rule carefully to close the lesser peritoneal cavity by attaching the margins of the opening in the transverse mesocolon to the stomach and not to the line of suture of the stomach to the jejunum or to the jejunum beyond the suture line.

Intestinal obstruction occurring three or four days after operation for acute perforative or suppurative appendicitis is sometimes difficult to differentiate from a secondary abscess with circumscribed peritonitis or a commencing diffuse peritonitis. The most reliable symptoms and signs in favor of obstruction alone, by which I mean obstruction not associated either with secondary or residual abscess or diffuse peritonitis, are: intermittent colicky pain with stormy peristalsis, inability to pass gas, persistent vomiting. If the obstruction is not relieved early, vomiting occurs, and is a regurgitation of the intestinal contents proximal to the obstruction, therefore the higher up the obstruction the earlier the regurgitant vomiting sets in. Often vomiting is accompanied by persistent hiccough due to absorption of toxins from the distended bowel. It is our practice in the Lankenau Clinic and in the Children's Hospital of the Mary J. Drexel Home to operate at once in the presence of these signs and symptoms. We have had one series of thirteen

cases of acute post-operative obstruction, all operated very early, with one hundred per cent. recoveries.

Paralytic distention of the bowel with regurgitant vomiting, with or without hiccough, and the absence of pain at all characteristic, so closely simulates actual obstruction or late peritonitis as to make the differentiation almost impossible except to the experienced surgeon, and even he may be in doubt.

The pathology usually is leakage of intestinal contents due to various causes, such as: ulcerative perforation of the appendix, or of a coil of bowel which at operation for strangulated hernia was thought to be viable enough to recover, and was returned to the peritoneal cavity; to the separation of a gastrojejunostomy or entero-enterostomy anastomosis; to the partial or complete opening of the duodenal stump after a subtotal gastrectomy; to escape of duodenal contents after excision and suture of a duodenal or gastric ulcer, or closure of a perforated ulcer, especially if a gastro-enterostomy has not been done; or leakage either of bile after a cholecystectomy; or of urine where the ureter has been accidentally incised, and in intraperitoneal rupture of the bladder.

When anastomoses are sutured throughout with catgut, leakage is more likely to occur on account of softening or dissolution of the gut before repair is advanced far enough to hold securely. I have had this occur, and therefore think it safer to use linen for the sero-muscular suture. In the case of an inverted duodenal stump or a sutured ulcer, if drainage has been used, recognition of the condition is easy and peritonitis is rarely diffused, but is only a circumscribed reparative peritonitis. Any of these occurrences calls for immediate operation which in my experience has saved lives that otherwise would have been sacrificed.

In any of these conditions the symptoms and signs are much the same. Some abdominal pain, rigidity, tenderness and slight distention, more or less general, but most marked at the site of lesion, vomiting, sometimes hiccough and inability to have a bowel movement or pass gas. As the condition advances the patient's expression becomes more and more indicative of a serious state of affairs.

I believe it is better to operate and not find an obstruction than to wait and then operate and find an obstruction that calls for an extensive resection, thus placing the case in the fifty per cent. mortality class. I cannot emphasize this too strongly. To wait until the abdomen is distended and silent means too often that the patient will also in a short time be silenced forever. On the other hand, the presence of distention and a silent belly often makes the diagnosis difficult. These are the cases that often are labeled peritonitis. In every case of acute obstruction there is a peritonitis, which in the early hours of the condition is limited, the exudate is merely serous, smears and cultures being negative for vicious bacteria. In these conditions therefore we work in a sterile field which is always a joy to the surgeon, while late operation calls for disentanglement of vicious entangling alliances, always a trial, whether in politics or in surgery. If in a recent post-operative case of

the acute abdomen, the patient while still on a liquid diet, develops cramp-like pain and nausea, I have the stomach washed out at once. If the washings have a foul odor, suspicious of upper intestinal contents, verified by immediate examination in the research laboratory, I operate at once. I have never regretted this course, while I have regretted having omitted it, especially when the family have insisted upon consultation with an internist or a gastro-enterologist. In my experience the delay has often spelled fatality. This is purely a surgical condition and not one for the physician who is not in daily association with the active surgeon delving into and solving the mysteries of intra-abdominal pathology in the living autopsy *in vivo*. It is in the operating theatre that the living teach the living, while in the laboratory of the dead house the dead teach the living.

Where the decision is that of post-operative peritonitis alone, the surgeon should stay his hands; and treatment, anatomic and physiologic rest, should be scrupulously carried out. I have not in any way been influenced by the different forms of treatment advocated for peritonitis, but hold steadfast to this measure. In our clinic, during 1925, there were 303 cases of acute appendicitis a large percentage of which were admitted with diffuse peritonitis with abscess, all of which were operated at the opportune time; the mortality was two per cent.

I have referred to making smears and cultures at the operations. This is done in every case operated on in the Lankenau Clinic. The report upon smears comes back in a few minutes after being taken, that of the cultures, in a few days. Incidentally I may say, the cultures verify the smears. The smears and cultures are taken of the fluid and the exudate, if present, as well as from the peritoneal field distal to the site of the lesion, and I may say, that I am largely influenced by the report of the smears in deciding upon drainage. In the Lankenau Clinic the laboratory corps coöperates with the surgeon; the Director of the Research Laboratory is always within call to assist the surgeon in settling certain pathological questions. This, I am sure, is of great advantage to our patients.

An occasional case of post-operative acute obstruction is attributable to the presence of drainage. I have never been sure that this was so, except in one instance where a glass tube having been used, a knuckle of bowel, the walls of which were thickened and in contact, was lapped around the tube. The proximal bowel was distended and the distal one collapsed. We must therefore accept the presence of drainage as a possible factor, so the less drainage the better. There is no doubt that the peritoneum does its best work in the absence of drainage, yet we cannot always dispense with it. Personally, I prefer heavy rubber dam for walling off, and rubber dam, rubber tube and occasionally cigarette drain for drainage.

Proper disposition and careful charting of drains is essential. They should not be placed so that a single knuckle of bowel is between them and the wall of the false or the true pelvis, nor should they occupy too devious a path in their exit. Abscess or pressure necrosis in the angle between the

ileum and the cæcum is very frequently a forerunner of an obstruction from plastic adhesions with kinking. I have so frequently found this condition that now in appendiceal abscess I often end the operation with an ileo-colostomy from an involved loop of ileum to the colon. At a first glance many theoretical objections can be urged against such a procedure. But experience and results outweigh logic and theory and I have not yet had any reason to change my practice. I know that such patients have a much smoother road to convalescence than was formerly the case. Occasionally when an obstruction is due to entanglements of the small bowel, one or more entero-enterostomies may be required.

Differentiation of post-operative paralytic ileus and obstruction from kinking is a most difficult problem. I find myself confronted with it very often and each time I attempt a solution I am full of anxiety. The life of the patient is frequently the price of an incorrect decision. As a rule, the situation arises on the third or fourth day after operation and is ushered in by increased abdominal distention, slight vomiting, a few mouthfuls at a time, with or without a putrid odor, often relieved for a time by gastric lavage; and shallow and rapid respirations. One is faced with the question: Is this a post-operative paralytic ileus, or is it due to obstruction by drains, kinking of the gut by exudate and adhesions, or to an organized abscess? It is impossible to give any rules or laws to govern either diagnosis or treatment. In perhaps no other condition does the solution so much depend on experience, judgment and surgical intuition. While sometimes the symptoms are due to peritonitis, not infrequently an obstruction is the basis and prompt relief means a living patient.

When I am not reasonably sure of the advisability of immediate operation, I institute the treatment of anatomic and physiologic rest, and if improvement does not take place in a few hours, I operate. I find that when the condition is due to peritonitis only, it usually clears up under this treatment, but not if obstruction is present.

Jejunostomy in paralytic ileus, in our experience, has not been satisfactory. In mechanical ileus jejunostomy, if made early, before changes in the bowel wall have taken place, serves a purpose, but even under such circumstances a sidetrack operation is preferable and in the absence of malignancy does not necessarily call for a further curative operation.

The diagnosis is most important. In fact, I wish to emphasize the fact that the diagnosis of acute obstruction is one of the most important pre-operative diagnoses we are called upon to make. The actual cause of obstruction is an excellent topic to discuss in the operating room while the surgeon scrubs up, but let it not be made a basis for delay of operation. One of the most potent causes of delay, as I have already said, is to be found in the indiscriminate and widespread use of morphine for acute abdominal pain. It is true that many cases of acute gastro-intestinal spasm may be permanently relieved by a hypodermic of morphia. It is also true that many cases of acute intestinal obstruction cannot in the first few hours be dis-

tinguished from such an attack. Every effort should be made to eliminate a mechanical obstruction to the bowel before resorting to morphine. But should a second or larger dose be indicated, mechanical obstruction should be considered a probability.

The earliest and most constant symptom of acute obstruction is *pain*, often extremely sudden and acute, at first colicky and intermittent, later continuous. It may be lulled to sleep by morphine, but reappears after the hypnotic effect has worn off, and often is worse than before. The next most common and constant sign is vomiting. This is frequent and copious, in fact, becomes more so the higher the obstruction. The function of the small intestine is mainly secretory, and this function seems greatly stimulated by the obstruction. The vomitus is at first gastric contents, then bile-stained fluid, finally stercoraceous. The fecal odor and character become more offensive as time elapses and are due to reverse peristalsis of the putrefying contents of the bowel above the obstruction. This stercoraceous vomiting is diagnostic of the terminal stage of obstruction, as are also the eructations of a few mouthfuls of the material every few minutes. In such cases operation promises little, if anything. The gastric lavage through a stomach tube, which should always be done before operating, is usually found to contain a large quantity of the offensive material, the removal of which gives the patient a great sense of relief and renders operation less likely to be accompanied by drowning in his own fluids. I have known the latter to occur where the stomach was not emptied before the anæsthetic was given. If at operation, in spite of the pre-operative lavage, the patient vomits regurgitant material, the stomach tube should be placed and left *in situ* during the operation. This minimizes the chance of the vomitus entering the larynx and running down into the bronchial tubes. The duodenal bucket will not always suffice as the fluid contents of the stomach are too thick to flow through the bucket.

In the very early stages of mechanical obstruction abdominal tenderness is absent. If the obstruction has existed for a day or two there is local tenderness corresponding to the site of the obstruction and due to the localized peritonitis which soon becomes generalized, followed by diffuse tenderness. The most important physical sign is hyperactive, stormy and whirring peristalsis up to the site of the obstruction and ceasing at that point. Later peristalsis becomes continuous, as do also the intestinal movements in their effort to overcome the obstruction. As toxæmia and peritonitis ensue, the character of the peristalsis changes to a tinkling sound, finally fading into the completely and ominously silent belly. At this stage the pulsation of the aorta is most marked, in fact is all that is heard on auscultation and is indicative of a grave condition.

A tumor is rarely felt except in cases of intussusception, nor is tympanitic distention very conspicuous until a later stage, except in cases of volvulus of the sigmoid. In these the distention is often enormous, and with the onset of peritonitis, greatly increases.

The characteristic sign in volvulus of the sigmoid is the enormous

meteorism which rises to the height of the fourth rib, seriously embarrassing respiration by crowding the diaphragm. This is almost diagnostic when combined with late or insignificant vomiting and early severe and often intermittent pain. Perforation is early and commonly due to delay. The other types of volvulus are so rare as to not permit of pre-operative differentiation. Unless this condition is recognized early and operated early, resection will be required. When the involved loop of sigmoid is not gangrenous, after untwisting the mesosigmoid, I have, in a few instances, successfully made a sigmoido-sigmoidostomy between the proximal and distal limbs at their origin. This not only gives relief but prevents recurrence of the volvulus.

Acute appendicitis is the type of acute abdomen that leaves most pathology in its trail. This is the meaning of the statement I have so often made, that the possibilities of acute appendicitis have no limitations. The pathology may consist of peritonitis, of coils of matted bowel causing obstruction, or of secondary abscess with obstruction, or residual abscess and obstruction; contraction of the walls of a cavity from which a large collection of pus has been evacuated and which causes angulation and obstruction of the wall of the bowel in contact with the cavity; adhesions, which in the course of their organization contract and ensnare coils of bowel resulting in obstruction; fecal fistula and obstruction; in addition there is the common, partial obstruction due to the infection having put the plexuses of Auerbach and Meisner out of commission.

The later effects of appendiceal pathological debris, occurring at various periods after the patient has recovered from the original operation are: intestinal obstruction from adhesions, obstruction due to rents of the omentum, and in some instances rents in the mesentery not observed at the primary operation and therefore not repaired, and ventral hernia. Obstruction occurring as a result of the last-named may be sub-acute or chronic.

Besides appendicitis, intestinal obstruction may be due to such conditions as tubercular peritonitis, tubercular inflammation of the mesenteric glands, infection of the pelvic viscera in the female, diverticulitis, agglutination of coils of bowel and obstruction, and very occasionally a cholecystitis which may result in adhesions and obstruction of the colon at the site of the hepatic flexure. Torsion of the great omentum and mesenteric thrombosis are among the rare causes of acute obstruction.

Although it is a generally accepted fact that the most common variety of acute obstruction in children is intussusception, in our experience in the Children's Hospital of the Mary J. Drexel Home, we operate more cases of obstruction following very severe appendicitis.

A word on intussusception. It is incomprehensible why so many of these cases come to operation with the bowel already gangrenous. I think it must be due to the false principle of trying medical treatment early in the case. As I view it, this is damnable, and often life destroying. The condition should be recognized early and operated at once. The death-rate would then be very small as against the present fifty per cent. mortality.

Obstruction may also be caused by an internal hernia. This type of hernia may occur through congenital or traumatic orifices in the diaphragm, usually on the left side. It is said that the gut may be ensnared by nine different kinds of duodeno-jejunal fossæ, as well as by the intersigmoid fossa, pericæcal fossa and the foramen of Winslow. These are all very rare and the diagnosis is practically never made before operation. Two years ago I operated upon a case in which two feet of jejunum had herniated through the foramen of Winslow, I reduced it successfully after much difficulty. The patient recovered and has remained well.

Intestinal obstruction from bands may result from any kind of local peritonitis, especially appendicitis, and tuberculous disease of the intestines or the intestinal glands. The bands may become stretched and rolled into cords by intestinal movements, and forming an arch beneath which the gut becomes ensnared; or a long band may form a loop in which the gut is knotted. A Meckel's diverticulum, or the remains of the vitello-intestinal duct, is present in about two per cent. of all subjects. Its end may be either free or attached to the umbilicus, mesentery, or any other point. It also may act as a band, the mechanism of which has already been given, or it may knot itself around a loop of gut if the end is free. It has been known to produce an intussusception or a volvulus.

Strangulated hernia is by far the commonest cause of acute intestinal obstruction. In any case where obstruction is suspected, the inguinal and femoral canals, the umbilicus, and the linea alba should be examined. If a hernia is discovered and is reducible, it usually can be ruled out as the cause of the obstruction, but whether it is irreducible, either temporarily or permanently, must be determined by extremely gentle pressure which should be attempted at once if the hernia is not tender or very tense. If forceful attempts are made to reduce a hernia there is always the possibility of damaging the bowel. Ulceration follows very quickly upon incarceration and even gentle pressure may empty the distended loop of bowel into the hernial sac.

As for taxis *versus* operation, the latter is by far the less dangerous. Even in the non-strangulated, irreducible hernia, taxis gives only temporary relief, and operation must finally be resorted to, to prevent a recurrence. In my experience taxis in strangulated hernia is more often responsible for a fatality than is failure to diagnose the hernia. Taxis is the cause of hemorrhage into the sac, into the mesentery, and into the wall of the bowel, thus precipitating early gangrene of the hernial contents. In the presence of fluid in the sac which occurs very early and soon becomes laden with colon bacilli, taxis exposes the peritoneum to the risk of infection by forcing some of the fluid into the peritoneal cavity. Reduction of hernia *en bloc*, without relieving the strangulation is another serious objection to taxis. When it was my mission to teach I always taught my undergraduate students not to make taxis. If this teaching were general, the operative mortality of strangulated hernia would be very much reduced. In operating a strangulated hernia, upon opening the sac it should immediately be thoroughly cleansed

before cutting the constriction, this provides a free communication with the peritoneal cavity through which the fluid will enter.

Strangulation may occur through slits and apertures, congenital or post-operative, the site being in the mesentery or omentum. Before sad experience proved the need of immediate repair, it was not uncommon to leave the rent in the transverse mesocolon after posterior gastro-enterostomy. A number of fatal internal hernias through this artificial aperture demonstrated this possibility in all operations where such rents are made, intentionally or otherwise, and led to their immediate repair.

Kinking is a very common form of acute obstruction due to dense adhesions, either post-operative, or following localized peritonitis. Several loops of bowel may be matted and kinked making an impassable lumen, or adhesions between the bowel and the peritoneum as well as of the pelvis, may form a V-shaped loop.

At operation, the surgeon, after making the incision must try to find the location of the lesion. In the absence of distention, this can often be done with the hand introduced and by touch, but the presence of distention precludes this manœuvre if the best interests of the patient are to be served. In the latter circumstances he looks for the collapsed bowel and follows the same to the site of the obstruction. This is attended by little or no risk of contusion or tearing the serosa or other damage to the bowel. When handling the distended bowel in the attempt to locate the point of obstruction, I expose the site of the lesion by disemboweling. In doing so, great care should be taken with regard to the proper disposition of the delivered coils of bowel and covering them with hot pads. Incidentally I may say that I rarely puncture or incise the distended bowel in order to empty it, because of the danger of infection; furthermore, I find, unless the case is a very late one, that when the obstruction is relieved, peristalsis of the distended coils is spontaneously reëstablished and emptying into the formerly collapsed bowel occurs. If the case is operated comparatively early, before there is evidence of decomposition, such as mottling, small hemorrhagic points in the walls of the distended coils, the bowel can be gently milked and the contents thus driven onward are kindly and promptly received by the collapsed coils. This is a part of the ritual of the operative technic. It is here that the sense of delicate touch is so essential to the operating surgeon if his results are to be crowned with success.

Acute obstruction may occasionally be due to the presence of a megacolon. Permit me to digress at this point in order to cite a case which emphasizes the importance of conservative as against radical surgery in the presence of a constipated megasigmoid in a child with presenting symptoms of acute obstruction. This boy presented a low abdominal tumor and obstruction. Opening the abdomen revealed a very large fecal tumor of a megasigmoid. The attending physician—one of Philadelphia's most prominent internists—rather insisted I make a resection. The boy not being very robust and not in any too good condition, I influenced the doctor to let me attempt emptying

the sigmoid by manipulation and the injection of warm sweet oil by rectum. I succeeded. The condition returned after two years, when I was again consulted. The patient being then in very fair condition, I operated and again was able to empty the constipated loop, and terminated the operation by a sigmoido-sigmoidostomy. Objection may be taken to this operation on the ground that the side-tracked loop may become distended with fæces and not being able to empty may ultimately ulcerate and perforate. I admit that this is one of the possibilities, but it has occurred only once in my experience. I have thought it possible for atrophy and loss of function to occur in the loop on account of the sigmoido-sigmoidostomy, which should be as large as possible, making a direct communication between the proximal and distal limbs of the offending loops.

This operation of course is not feasible in the presence of a gangrenous loop, but when the loop is in good or even fair condition, it should be considered. Resection of the acute sigmoid is always attended by considerable risk. Some of you may think a colostomy proximal to the questionable loop may be safer. This may or may not be so, and opens a question for discussion. In deciding the better operative procedure in the absence of gangrene, the blood-vessels of the meso-sigmoid must be free of thrombosis. In the presence of a questionable but not gangrenous condition of the loop, cæcostomy and the Mikulicz operation must also be kept in mind. These are questions that must be decided by the surgeon who, if his experience in dealing with acute obstruction is large, will know the right thing to do. I know of no class of cases requiring more mature judgment.

I later resected the loop which was much reduced in size and contained a very small amount of fecal matter. The last operation, from which the recovery was short and uninterrupted, was exceedingly well borne by the patient. He is growing rapidly into manhood and is perfectly well and entirely relieved of his constipation.

In chronic obstruction, except in the presence of an acute exacerbation supervening upon the same, operation need not be a hurry-up one. In the presence of an acute exacerbation, palliative treatment in the shape of anatomic and physiologic rest, lavage of the stomach, nothing in the way of medicine or nourishment by mouth, normal saline solution to which may be added glucose and whiskey by the Murphy drip method, ice bag to the abdomen, and enough morphia, hypodermically, to relieve the pain, will often be followed by subsidence of the acute condition when the case again becomes one of chronic obstruction. Chronic obstruction should be treated for a week or ten days before being subjected to operation. During this time study of the blood chemistry and renal function should be made and circulatory defects corrected or at least treated, if possible. The important pre-operative treatment is to secure a clear intestinal tract by the administration of mild purgatives, high enemata of sweet oil, etc., proper diet and in a few instances the making of a cæcostomy through which the bowel proximal to the obstruction can be irrigated and thoroughly emptied. I know of nothing

more disappointing and difficult to deal with than a loaded proximal bowel when making a resection or an anastomotic operation. It materially adds to the risk of infection, which means a peritonitis, or a faulty union, and if the patient survives, a fecal fistula. In fact, it may become necessary to make a fistula to save the patient's life.

Chronic obstruction often is carcinomatous in origin. The other causes are so rare that I will not discuss them. In most cases of chronic obstruction the history and careful physical examination will make the diagnosis. In obscure cases opening the abdomen alone will decide the question. X-ray study is important, but I do not consider it infallible by any means. In our experience in the Lankenau Hospital Clinic the diagnosis is usually made before the patient is referred for X-ray study. Visible peristalsis producing the ladder-rung abdomen is proof positive of the presence of a chronic obstruction. First rule out fecal obstruction. Clinically, it is worth bearing in mind, that in obstruction of the right half of the colon constipation is the rule, while if the obstruction is on the left, there is usually diarrhœa. There are exceptions to this, as there are to all rules. The procedure, that is, whether it is to be a one-stage or a two-stage operation, can usually only be settled after the abdomen is opened. The condition of the patient is also a guiding factor.

INTESTINAL OBSTRUCTION FOLLOWING APPENDECTOMY

STUDY OF TWENTY-ONE CASES

By Guy W. Carlson, M.D.
and
Victor F. Marshall, M.D.
of Appleton, Wis.

The frequency of post-operative obstruction, relatively speaking, is comparatively rare, but every surgeon who has access to a great many abdominal cases encounters a number of these occurrences. That most all cases are preventable, as claimed by some authorities, is rather too broad a statement. In spite of earlier diagnosis and definite improvement in operative technic, the mortality accompanying this condition is still very high. There are few surgical conditions which call for earlier operation than post-operative obstruction. If a certain stage is passed, as we can understand from the pathology of the condition, the removal of the obstruction does not save the life. Most authorities consulted give the mortality in operations for obstruction from 40 per cent. to 60 per cent.

Ochsner stated that nowadays most anyone attempts to perform an appendectomy and if the disease is in an early stage, his surgery results in recovery. The cases, however, that are more than a simple inflammation, offer a more serious problem. The end-results of these cases depends on the severity of the inflammation and especially on the judgment, experience and skill of the surgeon. In spite of due care and diligence, sooner or later one has to deal with cases of acute intestinal obstruction following acute appendicitis.

In reviewing the literature one is overcome with the meagerness of the discussion of this condition. It is one that should receive more careful consideration for it often occurs suddenly without any apparent warning, calling for immediate and definite interference.

Many cases of post-operative obstruction are due to adhesions, a large number have had drainage after appendectomy. If the appendix had been removed early, there would have been no formation of pus or resultant drainage, the amount of adhesions would have been limited and obstruction probably avoided. The general practitioner who usually sees the case first, must be aware of the importance of early operation, and therefore of early diagnosis. The education of the laity to the seriousness of such conditions would lead to earlier consultation.

Since January 1, 1924, approximately 875 operations for appendicitis have been performed at St. Elizabeth's Hospital of Appleton, Wis. From this group, we have collected fifteen cases of post-operative obstruction. In six additional cases the appendix had been removed prior to January 1, 1924. It is upon these cases that this paper is based.

The accompanying table is of interest from the standpoint of etiology and pathology.

TABLE I

Age	Sex	Findings at previous operation	Time between 1st and 2nd operation	Findings and prognosis.
22	M	Acute appendicitis; perforated	6 days	Spreading peritonitis intestinal adhesions—expired.
1	M	Acute appendicitis; perforated	20 days	Spreading peritonitis intestinal adhesions—expired.
35	M	Chronic appendicitis; gangrenous	3 years	Adhesions of ileum attached to scar—recovery.
52	M	Chronic appendicitis; gangrenous	8 years	Adhesions ileocæcal region attached to omentum forming twist of cæcum—recovery.
17	F	Chronic appendicitis	6 days	Definite Lane's kink—recovery.
54	M	Chronic appendicitis	12 years	Adhesions ileocæcal region attached to scar, a tense band from cæcum to ileum—recovery.
17	M	Acute appendicitis	8 years	Two linear bands, one from mesoappendix to ileum adhesions to cæcum attached to scar—recovery.
62	F	Acute appendicitis; perforated—abscess	18 years	Omentum adhered to scar, cæcum and attached ileum bound by adhesions—recovery.
8	F	Acute appendicitis; perforated—abscess	2 weeks	Twist of cæcum, adhesions cæcum to scar—recovery.
29	M	Chronic appendicitis; gangrenous	1 month	Agglutination ileum to ileum. Torsion of omentum. Omentum gangrenous adhered to scar—recovery.
33	M	Acute appendicitis; gangrenous—abscess	6 months	Agglutination ileum to cæcum, omentum adhered to scar—recovery.
46	M	Chronic appendicitis; perforated—abscess	12 days	Ileum adhered to scar, forming a kink—recovery.
14 mos.	M	Acute appendicitis; peritonitis	3 days	Intussusception of ileum, adhesions of mesoappendix—expired.
52	M	Chronic appendicitis; perforated—abscess	1 year	Omentum adhered to ileum causing constriction—recovery.
33	M	Chronic appendicitis; gangrenous	7 days	Adhesions of cæcum attached to scar—expired.
68	F	Acute appendicitis; perforated—abscess		Adhesions of cæcum attached to scar—recovery.
22	M	Acute appendicitis; gangrenous	2 months	Agglutination ileum to ileum, cæcum attached to scar—recovery.
30	M	Acute appendicitis; perforated—abscess	13 days	Agglutination ileum to ileum forming a kink—recovery.
43	F	Chronic appendicitis	7 months	Adhesions constricting ileum—expired.
7	F	Acute appendicitis; peritonitis	3 days	Volvulus or twist of ileum. Dense adhesions—expired.
26	M	Acute appendicitis; perforated—abscess	1 year	Many loops of intestine adhered to scar. Torsion of omentum—recovery.

In all of the cases the obstruction was caused by intestinal adhesions. The operative report of four cases revealed but slight involvement of the appendix at the first operation. These patients developed adhesions which later caused obstruction. It is seen that adhesions may form from operations of clean simple appendicitis as well as from those with marked inflammation, perforation or abscess formation. Perforation of the appendix occurred in eleven cases. In eight cases abscess formation was present. It is noted that in the cases in which the peritoneal cavity was invaded by microörganisms, adhesions had formed and obstruction taken place.

Previously existing suppurative conditions in the peritoneal cavity in some cases produced intestinal adhesions which later caused intestinal obstruction. Wound drainage in a number of cases was still present when discharged from the hospital. A spreading peritonitis occurred in four cases. In six cases a gangrenous appendicitis was present. In two instances preëxisting adhesions which were separated at time of operation leaving raw surfaces which could not be covered by peritoneum produced obstruction. Volvulus, too, caused a post-operative obstruction in two cases. The ileum in one case was drawn by a band of adhesions which gave rise to a pedicle over which a volvulus formed. In one instance adhesions caused a twist of the cæcum producing obstruction. In one case there was an intussusception of the ileum. In this instance a peritonitis was present.

There was a very decided difference in the two sexes; fifteen males (8.1 per cent.) compared with six females (2.8 per cent.).

TABLE II

What Was Done at Operation

	No. of cases
Separation of adhesions	18
Resection of bowel	1
Enterostomy or colostomy	2

Symptoms and Clinical Features.—The clinical picture was not always characteristic or constant. The onset of obstruction was varied greatly as to the time of occurrence following operation. The symptoms usually made their appearance during the first two weeks succeeding operation. Four of the cases had symptoms of post-operative obstruction during the first week. Nine cases developed obstruction during their stay in the hospital. Six cases had been discharged. The six remaining cases of post-operative obstruction had been operated for appendicitis prior to January 1, 1924 (Table I).

We must always bear in mind the possibility of an intestinal obstruction in our post-operative cases when there is present persistent vomiting, evidences of distention, peristaltic pain, increasing pulse rate, epigastric distress and a constipation that does not respond to gastric lavage and to enemas.

The onset was insiduous in most instances, passing gradually from the

post-operative state to that of acute obstruction. The early manifestations were peristaltic pain, slight watery regurgitation and vomiting, a distressed facies and constipation. The temperature in most instances did not go above 101 degrees F. The cases with a peritonitis usually had a higher reaction than those with abscesses. The pulse was accelerated in all cases but not as rapid as might be expected, except in those which were toxic. The white blood count was seldom higher than 20,000 and the polymorphonuclears higher than 90 per cent. The post-operative cases with abscess had the highest counts, spreading peritonitis somewhat less and those cases with symptoms of obstruction due to adhesions only had the lowest. The unperforated cases with adhesions likewise had a low white blood count. There was a complaint of fullness in the epigastrium with pressure symptoms and distress, shortness of breath which was somewhat relieved by belching, gastric lavage or vomiting. No rigidity of the abdominal muscles was present.

The later manifestations were stercoraceous vomiting or regurgitations of a brownish fluid with a fecal odor. The extremities became blue and cold. The abdomen became markedly distended, the pulse—rapid and feeble and later symptoms of collapse.

Prognosis.—In twenty-one cases there were six deaths, a mortality of 28.5 per cent. All cases (3) under four years of age had perforated and had a spreading peritonitis. These cases developed post-operative obstruction. Of the peritonitis cases (4) all died. In one case with peritonitis, an obstruction developed five days following operation. In this case a peritonitis was present. Of the remaining two deaths, adhesions produced complete obstruction. In both instances they had had recurrent attacks of appendicitis.

CONCLUSIONS

The outstanding fact brought out in this analysis is that an early diagnosis with operation reduces mortality, prevents development of complications, shortens convalescence, and makes sequelæ, such as post-operative intestinal obstruction less apt to develop.

The outstanding etiological factors were post-operative adhesions.

Adhesions causing intestinal obstruction may develop from operations of a simple clean appendicitis as well as from those with marked inflammation, perforation or abscess formation.

From a careful study of the records it is almost certain that a number of deaths attributed to "acute dilatation of the stomach," were secondary to intestinal obstruction. Such a diagnosis was made following operation for simple appendicitis.

The time interval between operation and the onset of obstruction is important. In this series obstruction occurred from two days to eighteen years following operation. A past history of appendicitis with drainage is an important factor in arriving at a diagnosis.

The three symptoms, peristaltic pain, vomiting and absolute constipation verified by the enema justify a diagnosis of post-operative obstruction.

The gentle handling of tissues, exclusion of peritoneal irritants, covering of the raw peritoneal surfaces and surgical cleanliness are important factors in the treatment. An important factor brought out in the history of many cases was the frequent use of cathartics. It would seem quite probable that cathartics are accountable for a large number of the early perforations which take place in children.

The prognosis is entirely dependent on early diagnosis followed by prompt surgical interference.

THE REDUCTION OF COLONIC INTUSSUSCEPTION BY AIR INFLATION

By Charles E. Farr, M.D.
of New York, N. Y.

Before the days of operative reduction of intussusception and even up to the present time, many cases of intussusception have been successfully reduced by means of enemata. It would seem safer to carry out this procedure than to do a laparotomy in very adverse circumstances, but in general the laparotomy is the only justifiable procedure, as one can never be sure of the success of pressure reduction unless he awaits the outcome. In such case it would be too late to intervene surgically if the attempted reduction proved unsuccessful.

There are several important advantages in the reduction by colonic pressure.

1. There is little or no shock.

2. The reduction is accomplished with great ease and rapidity, and with no danger whatever providing reasonable precautions are taken.

3. The force applied is much more effectual and much less likely to injure the already jeopardized bowel than manual reduction by laparotomy. The objection of course that it is a blind procedure with uncertain results is valid.

For this reason I have adopted the following treatment of intussusception, remembering that the great majority of these accidents occur in the colon. The child is prepared for laparotomy and the anæsthetic is begun. The nozzle of a Davidson syringe is inserted in the rectum and as the child begins to relax the colon is slowly inflated with air. This has a triple advantage, first, that it brings out of the pelvis and the left half of the abdomen a tumor which at times might otherwise escape palpation; second, that the elastic pressure of air is very little likely to damage the bowel; third, it escapes on removal of the syringe. The bowel is almost instantly emptied of its contents of air and the distention of the colon is entirely relieved.

The intussusception is now in the right iliac fossa. It may be, and frequently is, completely reduced. Theoretically at least, the toxæmia and shock from which the child has been suffering are relieved at once. Certainly the vascular supply of the bowel is immediately relieved of its impediment, except that part of the mass which may not have been completely reduced.

Careful palpation will reveal in many cases the remaining tumor in the right iliac fossa. This can sometimes be completely reduced with gentle pressure of the hands against the posterior abdominal wall. It would not be safe nor wise to consider the intussusception completely reduced unless a laparotomy is performed and the ileocæcal region carefully investigated. This is a matter of a few moments and can be done under local anæsthetic if

necessary. It will entail very little additional shock, and no operative procedure will be necessary within the abdomen, unless the apex of the intussusception is still caught. The reduction of this last bit is of course always the most difficult and dangerous part of the operation.

The points claimed then for the use of air inflation of the colon in such cases are that:

1. It lessens manual reduction after the abdomen is opened.

2. It immediately relieves the infant of at least a large part of the shock and toxæmia.

3. In certain obscure cases it renders a tumor palpable which otherwise might not be felt.

In illustration of the value of the points made, I submit certain recent cases:

Case I.—A six months' old infant was seen in private practice with a twenty-four hour history of intussusception. The history and physical findings were typical of this condition. Bimanual examination, finger in the rectum, and hand on the abdomen revealed a mass which was in the true pelvis, which was reduced to the ileocæcal region and there gently compressed for five minutes. This was without an anæsthetic. A laparotomy was immediately performed and the intussusception was found completely reduced. There was as is usual in such cases a marked œdema and infiltration of the appendix, but it was not removed. The wound was closed at once and recovery was uneventful.

Comment.—In this case a closed reduction was effected by bimanual examination. The method is crude and painful, but at times successful.

Case II.—An older child in the New York Hospital, service of Doctor Gibson, with a characteristic history of intussusception, but without the palpable mass. The bleeding in this case constituted a fairly serious hemorrhage from the bowel, and there was at the same time an extensive herpes of the left thigh and leg. Without an anæsthetic a thin barium mass was slowly injected into the colon. The head of the intussusception was quickly visualized and could be watched as it proceeded up the descending colon across the transverse colon, and down to the ileocæcal region. No attempt was made to complete the reduction manually. The child was immediately operated upon, the usual ileocæcal intussusception found and reduced without difficulty, and the child made an uneventful recovery.

Comment.—Here the barium mass was used instead of air for the purpose of diagnosis. It was of exceedingly great value in that respect, and it reduced nine-tenths of the intussusception.

Case III.—At St. Mary's Free Hospital for Children, a six months' old infant, sick forty-eight hours with the usual characteristic story, and an indefinite mass in the left lower quadrant. In this case under an anæsthetic the Davidson syringe was used, and the colon insufflated with air. The usual sausage-shape mass was felt definitely moving retrograde in the transverse colon, the hepatic flexure and eventually the ascending colon and cæcum. Here it disappeared during the examination. A laparotomy was at once performed, the intussusception found reduced entirely. There was marked congestion and œdema of the ileocæcal region and the appendix was almost black with blood oozing from the serosa. It was not removed. The abdomen was thoroughly explored. The wound was then closed. Convalescence was uninterrupted after the primary shock from the operation.

Comment.—In this case the complete reduction of the intussusception led me to fear that there might be another in some other portion of the bowel. Therefore, a complete examination, and much more pronounced shock.

Conclusions.—The internal pressure treatment of intussusception by enemata is an old and standard remedy, but a highly dangerous one unless laparotomy immediately follows the reduction. The use of air instead of water or other substance is urged because it is slightly safer, and it is more easily evacuated without soiling. The advantages of this combined method are obvious. 1. In diagnosis. 2. In completing a large part of the operation before the abdomen is opened, and occasionally a completion of the entire reduction.

One who has operated upon any considerable number of these cases knows that the manipulation of the mass intra-abdominally or its delivery on the abdomen, is a procedure beset with considerable danger and adds markedly to the shock. Anything which will simplify the procedure and lessen the shock should be readily adopted by the profession.

MECHANICAL FACTORS IN CHRONIC APPENDICITIS

By Hall G. Holder, M.D.
of San Diego, Calif.
and
William C. Menninger, M.D.
of Topeka, Kans.
from the second surgical (cornell) division of bellevue hospital, new york city

I. *Introduction.*—The material presented in this paper is derived from a statistical and follow-up study of 208 cases of chronic appendicitis treated on the Second Surgical (Cornell) Division of Bellevue Hospital. Only those cases were included in which the data were reliable and complete as to operative findings, pathological report, and well-defined history.

So much has been written concerning the many phases and theories of chronic inflammation of the vermiform appendix that it is difficult to segregate the pertinent comment. Much has been written about the errors of diagnosis of this condition. Even the existence of chronic appendicitis has been doubted because of the numerous failures to relieve symptoms by operation. Whiteford,[37] Kantor,[21] Connell,[4] MacLaren,[25] and others have pointed out a variety of conditions frequently mistaken for chronic appendicitis. Among these are included neurasthenia, stasis, and spasm in the cæcum and right colon, visceroptosis, anatomical anomalies of surrounding viscera, and associated disease of other pelvic organs. Morris [29] described what he called "irritative appendicitis," due to the normal involuntary process, in which the hyperplastic connective tissue presumably irritates the nerve-endings. He believes this causes symptoms which simulate chronic appendicitis and which are not relieved by operation. During the course of gynæcologic operations, Williams and Slater,[38] removed 500 appendices that had given no symptoms. They found one-third of this number to show lesions of a chronic nature described as chronic appendicitis. In one-half of this series the condition of the appendix could not be attributed to pelvic disease. Opposing this more conservative viewpoint are those of Gibson [12] and Gaither,[11] who emphasize the presence of a definite pathological and clinical entity in the form of chronic appendicitis with atypical symptoms often simulating ulcer, gall-bladder and kidney syndromes. However, they believe the appendix to be the seat of trouble and that removal relieves the symptoms, as shown by their careful follow-up records. Both of these observers indicate the necessity of careful pre-operative observation and also careful exploration at the time of operation to exclude any associated pathology of other organs regardless of the condition of the appendix.

Granting the existence of chronic appendicitis of an infectious, inflammatory nature, as well as the simulation of this syndrome by many non-associated factors, we believe it may also be produced or simulated by the action of certain mechanical factors, congenital or acquired, which affect the appendix and in some cases the adjacent gut.

TABLE I

Relation of Symptoms and Signs to Pathological Types

	Normal		Sclerotic		Catarrhal		Atrophic		Suppurative		Mucocele		Tuberculous		Oxyuris		Totals	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Total cases	48	23.0	113	54.3	16	7.6	16	7.6	9	4.3	3	1.4	3	1.4	2	0.96	208	
Males	26	12.5	64	30.7	8	3.8	8	3.8	6	2.8	3	1.4	3	1.4			118	56.7
Females	22	10.5	49	23.5	8	3.8	8	3.8	3	1.4					2	0.96	92	44.2
Recurrent symptoms	39	18.7	104	50.0	14	6.7	16	7.6	9	4.3	2	0.96	2	0.96	2	0.96	188	90.3
Constant symptoms	9	4.3	8	3.8	2	0.96					1	0.48	1	0.48			21	10.0
Pain right lower quadrant	42	20.1	96	46.1	13	6.2	15	7.2	8	3.8	3	1.4	3	1.4	2	0.96	182	87.5
Pain elsewhere	9	4.3	35	16.8	5	2.8	6	2.8	5	2.8	1	0.48					61	29.3
Tenderness right lower quadrant	44	21.1	88	42.3	13	6.2	9	4.3	8	3.8	2	0.96	2	0.96	1	0.48	167	80.2
Tenderness elsewhere	3	1.4	17	8.1	1	0.48	5	2.8	1	0.48	1	0.48	1	0.48			29	14.2
Tenderness rectal or pelvic	2	0.96	2	0.96	3	1.4			1	0.48			1	0.48			9	4.3
Nausea	27	12.9	61	29.3	12	5.7	12	5.7	7	3.3	2	0.96	2	0.96	2	0.96	125	60.0
Vomiting	15	17.2	51	24.5	6	2.8	10	4.8	7	3.3	1	0.48	2	0.96	1	0.48	93	44.7
Indigestion	3	1.4	9	4.3	2	0.96	1	0.48	1	0.48	1	0.48					17	8.1

Eructation	6	2.8	13	6.2	3	1.4	4	1.9									26	10.0
Anorexia	1	0.48	7	3.3													8	3.8
Constipation	26	12.5	38	18.2	7	3.3	5	2.8	4	1.9	3	1.4	3	1.4	1	0.48	87	41.8
Diarrhœa	1	0.48	2	0.96													3	1.4
Melena	1	0.48	2	0.96			1	0.48	1	0.48	1	0.48					5	2.8
Hæmatemesis			1	0.48			1	0.48									2	0.96
Jaundice	1	0.48	4	1.9	1	0.48											6	2.8
Dysmenorrhœa	4	1.9	3	1.4	2	0.95							1	0.48			10	4.8
Rigidity	9	4.3	10	4.8	4	1.9	4	1.9	1	0.48					1	0.48	29	14.2
Mass right lower quadrant	1	0.48	6	2.8	2	0.96					1	0.48			1	0.48	11	5.2
Distention	3	1.4	4	1.9	2	0.96	1	0.48					2	0.96			12	5.7
Admission temperature 98–98.9	27	12.9	60	28.8	7	3.3	3	1.4	2	0.96	2	0.96			1	0.48	102	49.0
99–99.9	11	5.2	18	8.6	1	0.48	6	2.8	3	1.4	1	0.96					40	19.2
100–100.9	2	0.96	10	4.8	2	0.96	1	0.48	3	1.4							18	8.6
101–101.9			1	0.48									1	0.48			2	0.96
102–102.9			1	0.48													1	0.48
103–over																	0	

II. *Symptomatology and End-results.*—Table I summarizes the occurrence of symptoms and signs associated with the different pathological conditions.

A careful study was not made of the sex and age incidence, since the surgical division has twice as many beds for male patients as female, which from the figures listed would indicate a preponderance of females. Deaver and Ravdin [7] in their series indicated that the actual percentage of females was higher. As only adult patients are included, no record of age incidence is made.

If the clinical interpretation of a "chronic appendix" is that of an appendix subjected to recurrent inflammation, the history of attacks of pain in the abdomen, especially in the right lower quadrant, is of significance. As the table indicates, the total number of patients having recurrent symptoms was 181, or 87 per cent. Of this number, 87 had an attack within one year, the remainder having longer histories. Deaver and Ravdin,[7] in their series of 500 cases, had 418 patients giving a history of previous attacks. In those histories in which the number of previous attacks of pain are recorded 32 had but one attack, 63 two attacks and 112 three or more preceding attacks. Fourteen patients complained of attacks of pain over a period of ten years; 52 stated the first attack had occurred from five to ten years before; 235 had attacks from one to five years ago, while only 83 had an attack within the year before admission. In our series 26, or 12.5 per cent., gave no history of a previous attack, complaining of a constant dull or burning pain in the right iliac fossa. This type of case occurred in 82 cases of Deaver's series (16.4 per cent.).

The outstanding symptom was periodic pain in the right lower quadrant. It occurred in 183 cases, or 87.9 per cent. In 63 cases, 30.2 per cent., however, the pain was located elsewhere in the abdomen, *i.e.*, in the epigastrium, right upper quadrant, and in a few instances in the left lower quadrant. Associated tenderness in the right lower quadrant was the next most frequently encountered symptom. It occurred in 168 cases, or 80.7 per cent. Tenderness was present elsewhere in 28, or 13.4 per cent. of the cases. Digestive tract disturbance was the third most frequent symptom. Constipation was present in 83, or 39.9 per cent., of the cases, nausea in 120, or 57.6 per cent., vomiting in 89, or 42.7 per cent., and eructations in 28, or 13.4 per cent. A normal temperature on admission occurred in the great majority of cases. Attention is called to the less frequent symptoms and signs listed.

In the 93 cases in which total leucocyte counts were made, the average count was 10,513, with extremes of 28,000 and 4000. The differential count as made in 87 of these cases, shows an average of 70.5 per cent. polymorphonuclears, with extremes of 87 and 47 per cent. The lowest average total white count occurred in the cases showing no microscopical pathology, being 8,968, and with also the lowest differential count, with 66.5 per cent. polymorphonuclears. A detailed study of the leucocyte counts in these cases is reported elsewhere.[27]

The end-results furnish us with data regarding the ultimate outcome, and are a direct check upon diagnostic and operative judgment. Definite follow-up records were complete in half the series as shown in Table II. Of this group there was complete relief in 81, or 77.1 per cent., partial relief in 16, or 15.2 per cent., and no relief in 8, or 7.6 per cent. The number and percentage for each pathological group are detailed in Table II. Pre-

TABLE II

End Results

		Normal	Sclerotic	Catarrhal	Atrophic	Suppurative	Mucocele	Tuberculous	Oxyuris	Totals
Complete	No.	18	51	8	3	3	1		1	81
Relief	%	17.1	48.5	7 6	2.8	2.8	0.9		0.9	77.1
Partial	No.	2	11	1		1				16
Relief	%	1.8	10 4	0.9		0.9				15.2
No	No.	2	4		2					8
Relief	%	1.8	3.6		1.8					7.6
No	No.	26	47	7	11	5	2	3	1	103
Follow-up	%	25.2	45.6	6.7	10.6	4.8	1.8	2.7	0.9	49.5
Pre-operative	No.	5	14	1				2		22
Complications	%	22.7	63.6	4.5				9.0		10.5
Mechanical	No.	47	88	13	12	9	3	1	2	
Factors	%	98.0	77.8	81.2	75.0	100.0	100.0	33.3	100.0	

operative complications occurring in the normal group included psychoneurosis, retroversion of the uterus, old Pott's disease, hemorrhoids and gastric ulcer. The one complication in the catarrhal group was gastric ulcer. The sclerotic group showed the following complications: Psychoneurosis, cirrhosis of the liver, pregnancy, pulmonary tuberculosis, syphilis, hyperthyroidism, epilepsy, gastric ulcer, cholecystitis, salpingitis, retroversion of the uterus. Two patients with tuberculous appendicitis had demonstrated pulmonary tuberculosis.

Two deaths occurred, or 0.95 per cent. of the total, one with complicating intestinal obstruction, the other with general peritonitis.

III. *Pathology.*—The division of the cases in this series is made on a pathological basis. In most instances the pathological department returned a careful description of the gross and microscopic picture with a diagnosis, making a fairly accurate classification possible. In addition the pathological report of the surgeon was available. The various groups are subsequently discussed under the mechanical factors.

Much discussion and difference of opinion have arisen concerning the morbid anatomy of chronic appendicitis in relation to the clinical manifestations. We have included two different ideas in the term chronic appendicitis: (1) That there may be a true chronic inflammation still in progress, or (2) the inflammation may have long since subsided, leaving a fibrotic process. Fibrosis with the production of a stricture or adhesions might well account for the symptoms. Often the appendix is free from adhesions showing only a progressive fibrotic process with gradual obliteration of the lumen. In the latter type of case it is difficult to see how symptoms are produced without the presence of a stricture. Although stricture formation is the result of a fibrotic replacement, essentially a pathological development, its effect on function is a mechanical one. It has therefore been included as a mechanical factor.

Regarding a generalized fibrosis in the structures of the appendix and its ability to produce the symptoms of appendicitis, there is a good deal of difference of opinion. Boyd [2] cites Aschoff as being very positive that obliteration from fibrosis is always due to previous inflammation. Ribbert, Zukerkandl, and others consider it a more or less natural atrophic and retrogressive process associated with the advance in years, and especially liable to occur in a vestigeal organ such as the appendix. Ribbert and Kelly, according to Boyd [2] and also the latter, himself observed in a large series that 25 to 33 per cent. of appendices removed at the post-mortem table and in the operating room showed obliterative changes. Williams and Slater,[38] in the routine removal of appendices in gynæcological operations, frequently found fibrotic changes in the appendices, with surrounding inflammatory changes in the form of adhesions, in the absence of symptoms of appendicitis. It is difficult to see why fibrosis in itself should give rise to symptoms, unless by a stricture, it causes obstruction or interference with the emptying of the organ. Fibrosis of the appendix, or as we have termed it, the sclerotic type, comprises the largest percentage of appendices in the pathological classification. As mentioned above, it frequently occurs without the slightest sign of appendicitis, suggesting the probability of associated factors which we will discuss subsequently.

On a microscopical basis the classification is as follows:

1. *Normal* appendices grossly show soft collapsible walls and histologically no pathological alteration. The explanation why such an appendix should produce symptoms, relieved by operation, we believe can be explained by associated mechanical factors.

2. *Sclerotic* changes in the appendix is manifested grossly by a rigid, thickened, incompressible tube, usually shorter than normal. Dilatation and tortuosity of the peritoneal vessels are common accompaniments. Frequently there are pale patches due to an obliterative process with surrounding adhesions and thickening of the mesentery, signifying a previous inflammatory process. On section the normal stellate appearance of the lumen is lost. The walls are round and thickened and the lumen is consequently narrowed.

Microscopically there is infiltration of all layers with new connective tissue obliterating the crypts and diminishing the lymphoid tissue in the mucosa. The submucosal connective-tissue zone is widened and there is deformity of the muscular coats by the fibrous infiltration. There is also thickening and deformity with increased density of the peritoneal coat.

3. The *Atrophic* type grossly presents the pale, withered appendix, which is firm, atrophic and shrunken. On section the appendix is seen to consist of two layers, an outer muscular layer, and an inner fibrous mass which includes what was once mucosa, submucosa and lumen. The lumen is usually completely obliterated. The microscopic picture shows an atrophic mucosa surrounded by a firm fibrous ring in the position of the submucosa which has come to include the inner atrophic muscular coat. The outer muscular coat is markedly atrophied and infiltrated with fibrous tissue while the peritoneal coat is thickened and deformed. Both this and the sclerotic type, the latter a more advanced picture of the same process, are the types generally considered to be typical of the chronic appendix. As mentioned above, considerable difference of opinion has been expressed as to its cause, *i.e.*, whether involutionary or inflammatory.

4. *Catarrhal* inflammation we have designated as the type affecting principally the mucous membrane of the appendix. In these appendices the gross appearance is little different from the normal. Occasionally they are slightly larger than normal. On section the mucosa is seen to be hypertrophied, with a consequent diminution in the size of the lumen and occasionally with a complete obliteration. Microscopically there is marked congestion and œdema of the mucosa. This condition may possibly resolve and undergo connective-tissue replacement, producing perhaps an early stage of a sclerotic appendix. In a large per cent. of this type, however, the increase in the size of the mucosa is seen to be due to a marked general hyperplasia of the lymphoid tissue, associated with status lymphaticus as detailed by Symmers and Greenberg [35] and by Miloslavich.[28]

5. *Suppurative* appendicitis, or acute diffuse appendicitis, is a generalized acute inflammation of this organ. Grossly the appendix is enlarged and thickened, the peritoneal coat reddened, and frequently there is an exudate on its surface. Subperitoneally, beginning abscess formation may be noted. On section all coats are swollen, the mucous membrane frequently ulcerated and the lumen filled with a muco-purulent material. Microscopically all coats are congested, œdematous, and infiltrated with polymorphonuclear leucocytes, frequently going on to abscess formation.

6. *Mucocele* of the appendix is described by Morrison [30] as a cystic dilatation due to obstruction of the appendix. There may be conversion of a diverticulum into a cyst in which the contents are thick and mucoid, or a true hydrops, in which the contents are watery. Frequently the mucoid material is collected into little balls which become calcified. Occasionally these cysts undergo malignant degeneration or rupture producing a condition of pseudomyxoma peritonei.

7. *Tuberculosis* of the appendix according to Warwick [36] may be primary or secondary, more usually the latter. Infection is hæmatogenous or lymphatic from surrounding organs that are involved. He states the symptoms closely resemble those of suppurative appendicitis, the diagnosis, however, most frequently being made microscopically. Scott [33] states that 0.5 per cent. of all appendices removed surgically are tuberculous.

TABLE III

Relation of Mechanical Factors to Pathological Types

		Normal	Catarrhal	Sclerotic	Atrophic	Suppurative	Mucocele	Tuberculous	Oxyuris	Totals
Mechanical	No.	47	13	88	12	9	3	1	2	175
Factors	%	98.0	81.2	77.8	75.0	100.0	100.0	33.3	100.0	84.1
Pelvic	No.	7	1	7					1	16
Position	%	14.5	6.2	6.1					50.0	7.6
Retrocæcal	No.	9	2	19	4	4	1	1		40
	%	18.7	12.5	16.8	25.0	44.4	33.3	33.3		19.2
Kink	No.	17	2	30	4	6			1	60
	%	35.3	12.5	26.5	25.0	66.6			50.0	28.8
Adhesions	No.	28	10	53	7	6	2		1	107
	%	57.2	62.5	46.9	43.7	66.6	66.6		50.0	51.4
Stricture	No.	8	4	30	6	3	1		1	53
	%	16.6	25.0	26.5	37.5	33.3	33.3		50.0	25.4
Adhesions	No.	12	1	20	1	2				36
about Cæcum	%	24.9	6.2	17.7	6.2	22.2				17.3
Adhesions	No.			9	2	1				12
about Colon	%			7.9	12.5	11.1				5.7
Fecaliths	No.	9		19	2					30
	%	18.7		16.8	12.5					14.4

8. *Parasitic Origin.*—The type associated with oxyuris vermicularis with the causation of appendicitis has been widely discussed. Fischer [10] found oxyuris in 110 routine appendectomies and in 28 per cent. at 105 necropsies, but could not determine a causal relationship. On the other hand, Harris and Donovan [16] found this worm in 22 cases out of an uninterrupted sequence of 121 appendectomies. He concludes from the pathologic condition of the appendices affected that these nematodes possess a definite rôle in the causation of appendiceal lesions. Armstrong [1] established it as a causative

factor in four cases. Riff [32] found oxyuris in 48 per cent. of 152 operative cases in adults and in 80 per cent. of 63 cases in children under fifteen years of age.

IV. *Mechanical Factors.*—Considerable has been written about the various mechanical factors found associated with chronic appendicitis. The causative rôle of these factors has been pointed out. In no instance to our knowledge has the subject been discussed from the standpoint of all the principle factors concerned. Dobbertin [9] states that in so-called chronic appendicitis one finds cases with pathology in the appendix and others with normal appendices. In the latter group, mechanical factors are the cause, most commonly attributed to adhesions about the ascending colon producing

TABLE IV

Per cent. Inter-relationship of Mechanical Factors

	Total number	No other mechanical factor	Pelvic	Retrocæcal	Kink	Adhesions	Stricture	Cæcal adhesions	Colon adhesions	Fecaliths
Adhesions	105	17.1	7.6	20.9	36.1		38.9	24.7	7.6	10.4
Stricture	62	11.2	4.8	17.6	40.0	65.6		19.2	6.4	17.6
Kink	62	12.8	6.4	27.3		61.1	40.2	11.2	8.0	12.8
Retrocæcal	40	17.5			42.5	55.0	27.5	20.0	7.5	10.0
Cæcal adhesions	35	5.7	8.5	22.8	19.9	74.1	31.3		11.4	11.4
Fecaliths	31	28.8	3.2	12.8	25.6	35.2	35.2	12.8	9.6	
Pelvic	16	18.7			25.0	50.0	18.7	18.7	12.5	6.2
Colon adhesions	13		15.4	23.1	38.5	61.6	30.8	30.8		23.1

kinks and stenosis with resulting appendicitis, colitis, pericolitis and even acute appendicitis.

An analysis of the mechanical factors found in the different types of appendices is presented in Table III. Some mechanical factors were present in over three-fourths of the cases in every group. In this series mechanical factors of some nature were found present in 98 per cent. of the normal group, 81.2 per cent. of the catarrhal, 77.8 per cent. of the sclerotic, and in 75 per cent. of the atrophic type. Mechanical factors were present in practically all of the suppurative appendices and in those with mucocele and oxyuris. This high percentage of mechanical factors found in an appendix which is microscopically normal suggests the possible etiology of the symptoms.

We have also drawn up a Table IV which shows the relationship of the several mechanical factors described. For example, of the 105 appendices described as having adhesions, 39.0 per cent. had strictures, 36 per cent. had kinks, 24.7 per cent. had cæcal adhesions, etc.

We propose to discuss below each type of mechanical factor found. For purposes of clarity we have retabulated the data for each type of mechanical condition. The figures below are all taken directly from Tables III and IV.

1. *Adhesions about Appendix.* Occurrence in one hundred and five cases.

Pathology (Table III)		Association with other Mechanical Factors (Table IV)	
Catarrhal	62.5%	No other mechanical factor	17.1%
Normal	57.2%	With stricture	38.9%
Sclerotic	46.9%	With kink	36.1%
Atrophic	43.7%	With cæcal adhesions	24.7%
Suppurative	66.6%	With retrocæcal position	20.9%
Mucocele	66.6%	With fecaliths	10.4%
Oxyuris	50.0%	With pelvic position	7.6%
		With colon adhesions	7.6%

Adhesions about the appendix which are responsible for abnormalities in position and other mechanical factors, such as kinks and strictures, are of unusual importance in this subject. Haberer [15] gave a detailed analysis of seven cases of chronic appendicitis the cause of which he believed was due to adhesions producing mechanical obstruction. Klauber [22] mentioned the causal relationship of mechanical factors to chronic appendicitis, citing particularly the formation of kinks by adhesions, with obstruction to the appendix, cæcum and ascending colon. In our series adhesions are well represented in all of the pathological groups. They occurred in over 50 per cent. of the cases in every group. Even in the normal group adhesions were present in 57.2 per cent. of the cases. The association of adhesions with abnormal positions of the appendix is not striking. However, the high percentage of adhesions occurring with both kink formation and stricture indicates a possible frequent explanation of the etiology of both. Adhesions about the cæcum were most commonly associated with adhesions about the appendix, in this series in 24.7 per cent. of cases.

2. *Adhesions about the Cæcum.* Occurrence in thirty-five cases.

Pathology (Table III)		Association with other Mechanical Factors (Table IV)	
Normal	24.9%	No other mechanical factors	5.7%
Suppurative	22.2%	With adhesions about appendix	74.1%
Sclerotic	17.7%	With stricture	31.3%
Catarrhal	6.2%	With retrocæcal position	22.8%
Atrophic	6.2%	With kink	19.9%
		With colon adhesions	11.4%
		With fecaliths	11.4%
		With pelvic position	8.5%

Jackson [18] was the first to mention the symptom complex simulating chronic appendicitis produced by adhesions about the cæcum and ascending colon. He operated on nine cases in one year in which the condition was relieved by the freeing of these bands. Mayo [26] describes the membranes about the cæcum as embryonic in origin. He believes they are due to late

rotation of the bowel and descent of the cæcum from its hepatic position after the formation of the parietal portion of the peritoneum. Jacobson [19] and Brown [3] emphasize the rôle of fecal stasis in the cæcum and ascending colon, produced by bands and adhesions, resulting in a low-grade inflammatory process causing symptoms of chronic appendicitis.

In but 5.7 per cent. of our cases were there no other associated mechanical factors, yet 24.9 per cent. of normal appendices showed these adhesions suggesting the possible causative rôle in the production of symptoms. The fact, too, that the retrocæcal position occurred associated in 22.8 per cent. of cases suggests these adhesions as a likely factor in its production. The presence of a kink in approximately 20 per cent., and stricture in 31 per cent. is noted. The presence in 74.1 per cent. of cases of adhesions about the appendix is the largest for the series, the regional reaction suggesting either a surrounding low-grade inflammatory process or possibly a congenital condition.

3. *Adhesions about the Colon.* Occurrence in thirteen cases.

Pathology (Table III)		Association with other Mechanical Factors (Table IV)	
Atrophic	12.5%	No other mechanical factor	0.0%
Suppurative	11.1%	With adhesions about the appendix.	61.6%
Sclerotic	7.9%	With kink	38.5%
		With stricture	30.8%
		With cæcal adhesions	30.8%
		With fecaliths	23.1%
		With retrocæcal position	23.1%
		With pelvic position	15.4%

Harvey [17] examined 105 infants and demonstrated the frequent occurrence of attachments of the colon, appendix and terminal ileum. These variations he believed identical with certain adhesions seen in the adult about the gall-bladder, hepatic flexure, ascending colon ("Jackson's membranes"), cæcum, appendix and terminal ileum ("Lane's band"), and frequently but wrongly ascribed to inflammation. These, he believed, had a demonstrated relation to the syndrome of chronic appendicitis. Gregoire [14] says that the vascular membranes that develop enclose the colon in a sac that is too short for it and the colon consequently has to bend. If a congenital origin is accepted, then it may be assumed that the colon has been impeded in its growth. The colon thus folded upon itself permits of obstruction of its contents with a symptom complex typical of chronic appendicitis. Shutt [34] states that pericolic adhesions form a definite pathological entity and frequently exist without any trouble with the appendix. Simple removal of the appendix in these cases does not afford relief. Davisons and Royer [6] have described a definite surgical entity with symptoms of chronic appendicitis associated with the presence of marked deforming bands and adhesions in the region of the ascending colon. They believe them to be produced by a low-grade inflammatory process induced by colon stasis. Chronic appendicitis is sometimes an accompanying condition, but is not the causative factor. This condition is readily diagnosed,

they state, by means of röntgenographic examinations and its relief obtained by surgical interference, correcting this definite mechanical condition.

In our series the frequency with which colon adhesions are associated with the retrocæcal position, 23.1 per cent., suggests that they may be a causative factor in the latter. Also the high per cent. of association with kink, stricture and adhesion formation about the appendix is noted. In no instance did adhesions about the colon occur alone without other mechanical factors.

4. *Kink of the Appendix.* Occurrence in sixty-two cases.

Pathology (Table III)		Association with other Mechanical Factors (Table IV)	
Normal	35.3%	No other mechanical factors	12.8%
Sclerotic	26.5%	With adhesions about the appendix.	61.1%
Atrophic	25.0%	With stricture	40.2%
Suppurative	66.6%	With retrocæcal position	27.3%
Oxyuris	50.0%	With fecaliths	12.8%
Catarrhal	12.5%	With cæcal adhesions	11.2%
		With colon adhesions	8.0%
		With pelvic position	6.4%

De Forest [8] points out the abnormal shapes encountered due to the kinking which produces disturbances in circulation and which accounts for the intermittent symptoms in chronic appendicitis. Klose [23] described torsion of the appendix as a factor simulating chronic appendicitis. Jones-Evans [20] frequently observe various degrees of torsion usually in the direction from left to right, the mesentery becoming wrapped around it. The torsion varies from a slight twist to complete strangulation, which on progression produces intermittent symptoms simulating recurrent attacks of chronic appendicitis.

It is noteworthy that in 27.37 per cent. of cases kink was associated with the retrocæcal position bearing out the previous discussion of the possible mechanical association. The high percentage of adhesion formation about the appendix would be expected in the production of this factor. The occurrence of the associated stricture formation in 40.25 per cent. of cases, higher than for any other group, would signify that by either mechanical or irritative means kinking promotes the production of fibrous tissue with stricture formation. Discounting the groups represented by only a few cases, this factor occurred most frequently in the normal appendix group (35.3 per cent.). It is to be noted also that six of the nine cases in the suppurative group showed a kink, the latter possibly being responsible for complete obstruction of the lumen and decomposition of the retained contents. Infection of the mucosal wall presumably occurs, finally going on to suppuration.

De Forest,[8] and especially Pitzman,[31] call attention to stricture formation in the causation of chronic appendicitis. Pitzman believes that all appendicitis, acute or chronic, depends upon the formation of a stricture, the latter developed probably from a primary simple ulcer. Attacks of acute suppurative appendicitis he believes are brought on by the complete closure of a preformed stricture with resulting fecal stasis, infection of the appendiceal

5. *Stricture of the Appendix.* Occurrence in sixty-two cases.

Pathology (Table III)		Association with other Mechanical Factors (Table IV)	
Atrophic	37.5%	No other mechanical factor	11.2%
Sclerotic	26.5%	With adhesions about appendix	65.6%
Catarrhal	25.0%	With kink	40.0%
Normal	16.6%	With cæcal adhesions	19.2%
Suppurative	33.3%	With fecaliths	17.6%
Mucocele	33.3%	With retrocæcal position	17.6%
Oxyuris	50.0%	With colon adhesions	6.4%
		With pelvic position	4.8%

wall, reflex pain, temperature and leucocytosis. The true chronic appendicitis also has a stricture, which, however is patent during intervals between attacks, the resultant pathology being due to the previous inflammation and perhaps the continuance of a low-grade inflammatory process.

As would be expected, stricture formation would be most frequently associated with a fibrotic, obliterative process as in the atrophic type associated in this series in 37.5 per cent. of cases. Its association with a high percentage of kink and adhesion formation, 40, and 65.5 per cent., respectively, would suggest a causal relationship. The high percentage, 17.6 per cent., of associated fecalith formation is of interest. The obstructive action of the stricture probably causes the inspissation of retained fecal material.

6. *The Retrocæcal Position.* Occurrence in forty cases.

Pathology (Table III)		Association with other Mechanical Factors (Table IV)	
Atrophic	25.0%	No other mechanical factor	17.5%
Normal	18.7%	With adhesions about appendix	55.0%
Sclerotic	16.8%	With kink	42.5%
Suppurative	44.4%	With stricture	27.5%
Tuberculous	33.3%	With cæcal adhesions	20.0%
Mucocele	33.3%	With fecaliths	10.0%
Catarrhal	12.5%	With colon adhesions	7.5%

Gladstone and Wakely,[13] in observing the position of the appendix in three thousand necropsies, have classified them as follows:

Anterior or pre-ileal	27 cases	0.9%
Splenic of post-ileal	15 cases	0.5%
Pelvic	828 cases	27.5%
Subcæcal	56 cases	1.8%
Post cæcal and retrocolic	2076 cases	69.2%
Ectopic	1 case	0.03%

Crabb,[5] in discussing the above figures, points out that in the embryological development of the appendix it may take many various positions depending on the degree of rotation at the ileocolic junction. This, however, does not explain why 70 per cent. of appendices occur in the retrocolic or retrocæcal position. Crabb does not believe this to be the normal position, although it is preponderantly found here. He states that because of this position, the appendix is more apt to become inflamed and consequently comes to our attention more frequently. He believes the appendix is more likely to become

diseased in this position because of impaired circulation or actual obstruction produced when the cæcum is filled.

The frequent occurrence in our series of the retrocæcal position with kink and adhesion formation may be of some significance. Since the retrocæcal position is prone to produce kinking at the base with obstruction, especially when the cæcum is dilated, this high frequency of kink formation might be expected. The associated presence of adhesions about the cæcum in 20 per cent. of cases is also of interest concerning its formation and maintenance. It was found that the pathological groups most commonly affected were the atrophic where it occurred in 25 per cent. of cases and in the normal group in 18.7 per cent. of cases.

7. The Pelvic Position. Occurrence in sixteen cases.

Pathology (Table III)		Association with other Mechanical Factors (Table IV)	
Normal	14.5%	No other mechanical factors	18.7%
Sclerotic	6.1%	With adhesions about appendix	50.0%
Catarrhal	6.2%	With kink	25.0%
Oxyuris	50.0%	With cæcal adhesions	18.7%
		With colon adhesions	12.5%
		With stricture	18.7%
		With fecaliths	6.2%

This position was second in frequency in the series of Gladstone and Wakely detailed above. In our series it was associated with no other mechanical factors in 18.75 per cent. of cases having symptoms of chronic appendicitis. Of these 14.5 per cent. were classified by the pathologist as normal appendices. Because of its position it seems possible that many factors might operate to disturb blood supply and interfere with the normal evacuation of its lumen, perhaps thereby accounting for the initiation of the pathological processes and symptoms. This position permits it to encroach on adjoining pelvic viscera and exposes it to involvement by pathological processes occurring in them.

8. Fecaliths of the Appendix. Occurrence in thirty-one c

Pathology (Table III)		Association with other Me Factors (Table IV)	
Normal	18.7%	No other mechanical facto s	28.8%
Sclerotic	16.8%	With adhesions about appendix	3[illegible] %
Atrophic	12.3%	With stricture	35. %
		With kink	25.0%
		With retrocæcal position	12.8%
		With cæcal adhesions	12.8%
		With colon adhesions	9.6%
		With pelvic position	3.2%

In a personal communication Kummel [24] states the following as his opinion: "That the appendix because of its anatomically unfortunate cul-de-sac form must give rise to frequent stasis of fecal particles and secretions is self-evident. The insult to the appendix of retained fecal material one can

readily see in going through a large number of accurate case histories, the symptoms often dating back to early childhood. I have been able in countless preparations to follow the pathological changes, increasing more and more in the appendix. Entrance of fecal matter, its escape, and then stagnation of the same, swelling of the mucosa, formation of small fecaliths, enlargement of these, ulceration of the mucosa, formation of strictures, kinking and then adhesion formation. All of the last-named factors are secondary to retention of fæces with fecalith formation in the appendix with secondary erosion and scar tissue formation in the form of a stricture. No normal appendix contains fæces. If such is found then an early nidus for pathological changes is present."

The high per cent. of cases in which fecaliths occurred alone in our series, to account for the symptoms is a noteworthy fact. The high frequency of association with a kink, adhesions about the appendix and a stricture is expected in view of the supposed knowledge concerning their formation from obstructed fecal material.

V. CONCLUSIONS

(1) A large per cent. of the cases commonly diagnosed "chronic appendicitis" are directly caused by a variety of mechanical factors acting on the appendix. These may be congenital or acquired in origin, but either type may affect blood supply and motility leading to pathological changes and symptoms. Such cases might be more correctly termed "mechanical appendicitis."

(2) Appendices which were microscopically normal presented some mechanical abnormality in 98 per cent. of cases. Hence an inflammatory process is not a prerequisite to the clinical picture of "chronic appendicitis." When present, it is secondary to a previously abnormal mechanical status of the appendix.

(3) Adhesions, either about the appendix, cæcum, or colon are the most common mechanical abnormality, occurring in 73.5 per cent. of the series. The frequent association with other mechanical factors is possibly secondary to the adhesio[illegible].

(4) Kink of [illegible]ppendix occurred most frequently in the normal appendix (3[illegible]3 per ce[illegible]d was commonly associated with the retrocæcal position, adhesion and [illegible]tricture formation.

(5) [illegible]icture formation occurred in nearly a third of all cases, most commonly with the atrophic type of appendix (37.5 per cent. of cases). In two-thirds (65.5 per cent.) it was associated with adhesions.

(6) The retrocæcal position exercised a definite causative rôle in the production of symptoms. It occurred in 40 cases, in 18.7 per cent., of which the appendices were normal. In 17.5 per cent. there were no other mechanical factors. Probably as the direct result of position and the mechanical action of a dilated cæcum, a kink occurred in 42.5 per cent. of these cases.

(7) The rôle of fecal stasis as a causative factor is probably underestimated. Fecaliths occurred in 31 cases in this series, in 28.8 per cent. of which

they alone were present to account for the symptoms. It is very likely that in many cases, the inflammatory process follows infection from fecal stasis in the appendix, although definite fecal concretions are not present. Frequent association of fecaliths with stricture formation (35.2 per cent. of cases) and kink (25.6 per cent.) is to be expected in view of the known method of their formation.

(8) End-results show appendectomy benefited these patients. In the group showing no pathological changes, with 98 per cent. complicating mechanical factors, 22 cases are represented, 20, or 91 per cent., being benefited by removal of the appendix while 2, or 9 per cent., experienced no relief. Of the 105 cases in which there were accurate follow-up data, 97, or 92.4 per cent., were relieved by operation. Eight, or 7.6 per cent., were not helped.

We wish to express our appreciation to Dr. Harold E. Santee, Director of the Second Surgical Division, for his courtesy in affording us the opportunity of reporting the above cases.

REFERENCES

[1] Armstrong, A. W.: Pin Worms as a Cause of Appendicitis. New York State Journal of Medicine, vol. xvi, p. 470, September, 1916.

[2] Boyd, W.: Surgical Pathology. W. B. Saunders Company, 1st Edition, 1925.

[3] Brown, T. R.: Visceroptosis and Chronic Appendicitis. Medical Clinics of North America, vol. i, p. 189, July, 1917.

[4] Connell, F. G.: Pseudo-appendicitis. J. A. M. A., vol. lxvii, p. 335, July 29, 1916.

[5] Crabb, G. M.: The Anatomy of the Appendix as Related to Acute and Chronic Appendicitis. Journal of Iowa State Medical Society, vol. xv, pp. 69–72, February, 1925.

[6] Davison, C., Davison, M., and Royer, D. J.: Adhesions about the Ascending Colon Simulating Chronic Appendicitis. Surgery, Gynecology and Obstetrics, vol. xxxviii, pp. 171–180, February, 1924.

[7] Deaver, J. B., and Ravdin, I. S.: End Results of Five Hundred Cases of Chronic Appendicitis. Arch. of Surg., vol. vi, pp. 31–40, January, 1923.

[8] De Forest, H. P.: Causes of Appendicitis. N. Y. Med. Jour., vol. cvi, pp. 501–505, September 15, 1917.

[9] Dobbertin, E.: Zur Pathogenes der sogen. Chronischen Blinddarment-gundung. Deutsch. med. Wchnschr., vol. xxxix, pp. 1549–1551, August 7, 1913.

[10] Fischer, W.: Deutsche Zeitschriff fur Chirurgie, vol. clxxxiii, pp. 129–287, November, 1923. ab. Oxyuris and Appendicitis. J. A. M. A., vol. lxxxii, p. 829, March, 1924.

[11] Gaither, E. H.: Chronic Appendicitis. The Southern Med. Jour., vol. xiv, pp. 190–199, March, 1921.

[12] Gibson, C. L.: The Results of Operations for Chronic Appendicitis. Am. Jour. Med. Sciences, vol. clix, p. 654, May, 1920.

[13] Gladstone, R. J., and Wakeley, C. P. G.: The Relative Frequency of the Various Positions of the Vermiform Appendix. Brit. Jour. Surg., vol. ii, pp. 503–529, January, 1924.

[14] Gregoire, R.: Membranous Pericolitis and Chronic Appendicitis. Archives des Maladies de l App. Digestif, Paris, vol. iii, p. 10, May, 1919. abs. J. A. M. A., vol. lxxiii, p. 299, July, 1919.

[15] Haberer, H.: Appendicitis Chronica Adhæsiva. Mitt a. d. Grenz. geb. d. Med. u. Chir., vol. xviii, pp. 169–194, 1907.

[16] Harris, W. H., and Donovan, B. C.: Oxyuris Vermicularis as a Causative Factor in Appendicitis. J. A. M. A., vol. lxxxiv, pp. 650–654, Februarv 28, 1925.

[17] Harvey, S. C.: Congenital Variations in the Peritoneal Relations of the Ascending Colon, Cæcum, Appendix and Terminal Ileum. ANNALS OF SURGERY, vol. lxvii, pp. 641–686, June, 1918.

[18] Jackson, J. N.: Membranous Pericolitis. Surg., Gyn. and Obst., vol. ix, pp. 278–287, September, 1909.

[19] Jacobson, J. H.: The Chronicity of Appendicitis. Am. Jour. Obst. and Gyn., vol. lxvii, pp. 493–500, March, 1913.

[20] Jones-Evans, E. J. L.: Mechanical Factors in the Causation of Appendicitis. Practitioner, vol. cxiv, pp. 113–180, February, 1925.

[21] Kantor, J. L.: On Five Common Clinical Types of Appendicitis. Med. Clinics of North Am., vol. v, pp. 1081–1112, January, 1922.

[22] Klauber, O.: Die Aetiologie der Appendizitis. Munch. med. Wchnschr., vol. lvi, pp. 451–455, March 2, 1909.

[23] Klose, H.: Die Habiturlle Torsion des mobilen Zoekum. Ein typisches Krankheitsbild. Munch. med. Wchnschr., vol. lvii, pp. 348–350, February 15, 1910.

[24] Kummel, H.: Personal Communication, November, 1925.

[25] MacLaren, A.: Chronic Appendicitis and It's Relation to Visceroptosis. ANNALS OF SURGERY, vol. lxiv, p. 579, November, 1916.

[26] Mayo, C. H.: Intestinal Obstruction Due to Kinks and Adhesions of the Terminal Ileus. Surg., Gyn. and Obst., vol. xii, pp. 227–230, 1911.

[27] Menninger, W. C., and Holder, H. G.: The Relation of Total and Polymorphonuclear Leukocyte Counts in Chronic Appendicitis. Accepted for Publication in ANNALS OF SURGERY.

[28] Miloslavich, E.: Zur Pathogenese des Appendizitis. Wien Klin. Wchnschr., vol. xxv, pp. 442–443, 1912.

[29] Morris, R. T.: Notes on Four Kinds of Appendicitis. ANNALS OF SURGERY, vol. lxvi, p. 560, November, 1917.

[30] Morrison, W. P.: Fish-Egg Mucocele of the Appendix—A Cystic Type of Chronic Catarrhal Appendicitis, with Report of a Case. Boston Med. and Surg. Jour., vol. clxxxviii, p. 532, April, 1923.

[31] Pitzman, M.: No Surgical Appendicitis without Organic Stricture. ANNALS OF SURGERY, vol. lxxi, pp. 149–154, February, 1920.

[32] Riff, A.: Etiology of Appendicitis, abs. J. A. M. A., vol. lxxiii, p. 1477, November, 1919. Presse Medicale, Paris, vol. lii, p. 27, September 18, 1919.

[33] Scott, J. R.: Tuberculosis of the Appendix. ANNALS OF SURGERY, vol. lxvi, pp. 648–653, December, 1917.

[34] Shutt, C. H.: Pericolic Adhesions as a Factor in Diagnosis of Chronic Appendicitis and Failure to Obtain Relief by Removal of Appendix. Missouri State Med. Asso. Jour., vol. xxii, p. 249, July, 1925.

[35] Symmers, D., and Greenberg, M.: The Clinical Significance of Lymphoid Hyperplasia of the Appendix. J. A. M. A., vol. lxxii, pp. 468–470, February 15, 1919.

[36] Warwick, M.: Tuberculosis of the Appendix. ANNALS OF SURGERY, vol. lxxi, p. 139, February, 1920.

[37] Whiteford, C. N.: The Chronic Appendix. Practitioner, vol. cix, pp. 155–165, August, 1922.

[38] Williams, J. T., and Slater, R.: The Condition of the Appendix in Five Hundred Laparotomies on Patients Representing no Symptoms of Appendicitis. ANNALS OF SURGERY, vol. lxx, pp. 535–538, November, 1919.

STABILIZATION OF PARALYTIC TALIPES VARUS*

By Francis S. Chambers, M.D.

of Philadelphia, Pa.

Talipes varus, due to paralysis of the peroneal muscles, is essentially an exaggeration of the "inversion twist" carried beyond the limits of normal joint motion by the action of abnormal muscle pull. It consists of a series of important distortions which seriously disturb the alignment of the foot. In the more severe types of the deformity, these distortions are usually fixed by adaptive changes in the osseous structures of the foot. When these changes are well developed, the deformity can seldom be completely corrected unless the bones are redressed to proper shape.

The sub-astragalar and "Chopart" joints are the "key" to the correction of the deformity and should be exposed to the vision of the operator. It is then an easy matter to trim the osseous irregularities as indications demand and obtain an anatomical restoration which will be lacking only in flexibility over which there is insufficient muscular control. The remodeled foot should be placed in alignment with the ankle-joint.

The operation which I employ is based on these principles. The technic is by no means difficult but an intimate knowledge of the mechanism of the deformity is essential. It is not to be regarded as a "panacea" for all forms of talipes but should be reserved for varus deformities which are caused by paralysis of the peroneal muscles.

Anatomical Considerations.—The deformity consists of five important distortions, four of which are elemental to the "inversion twist" of the foot. They take place beneath the astragalus. The ankle-joint is everted owing to the external torsion of the lower leg.

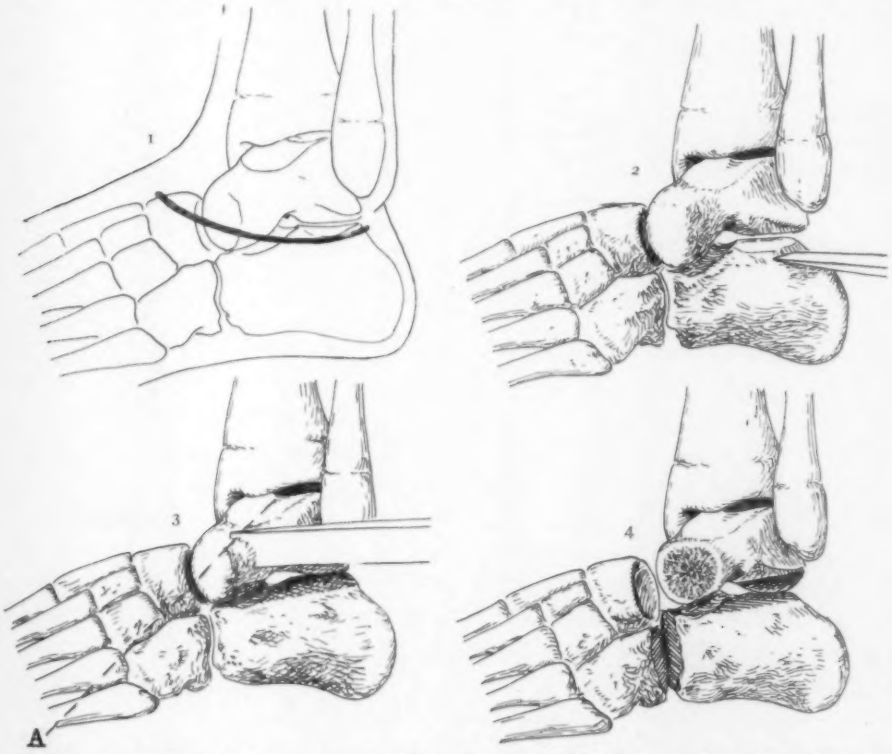
1. External torsion of the lower leg with eversion of the ankle-joint. In connection with this distortion, the external malleolus is displaced backward and forms a conspicuous prominence on the outer side of the ankle. Its posterior displacement is due to paralysis of the peroneal muscles. Normally, these muscles play an important rôle in moulding the malleolus during infancy and early childhood. With each contraction, their tendons exert a forward thrust upon the malleolus, thus maintaining it in proper relation to the ankle-joint as growth progresses. If this moulding force is lost at an early age through paralysis of the peroneals, the malleolus will remain "slumped" posteriorly, and the earlier this loss occurs the greater will be its posterior displacement.

The internal malleolus is in turn shifted further forward by the influence of several factors, one of which is probably unbalanced muscle pull. As a result of the altered relation of the malleoli, the ankle-joint is everted, the

* Read before the Philadelphia Academy of Surgery, May 3, 1926.

direction of its motion is deviated obliquely outward and forward in relation to the leg.

These changes constitute an important distortion. Hoke (*Journal of Orthopedic Surgery*, Oct., 1921, vol. xix, No. 10) has emphasized its importance referring to it as "external torsion of the lower tibia." It is a characteristic feature of talipes varus due to peroneal paralysis which develop in infancy or early childhood. It is usually present in neglected cases of



FIGS. 1 to 4.—Steps of the operation. Drawings constructed from X-ray tracings of a case of paralytic varus. 1. Incision. 2. Resection of sub-astragalar joints. 3. Excision of the head of the astragalus by an oblique osteotomy. The chisel cut is made at a right angle to the direction of ankle motion (dotted line "A"). Owing to the eversion of the ankle-joint, the osteotomy is oblique in relation to the foot. 4. Sub-astragalar joints resected, head of astragalus has been excised by oblique osteotomy, calcaneo-cuboid joint also resected. The "adduction beak" has been removed thus "squaring" the forward end of the os-calcis.

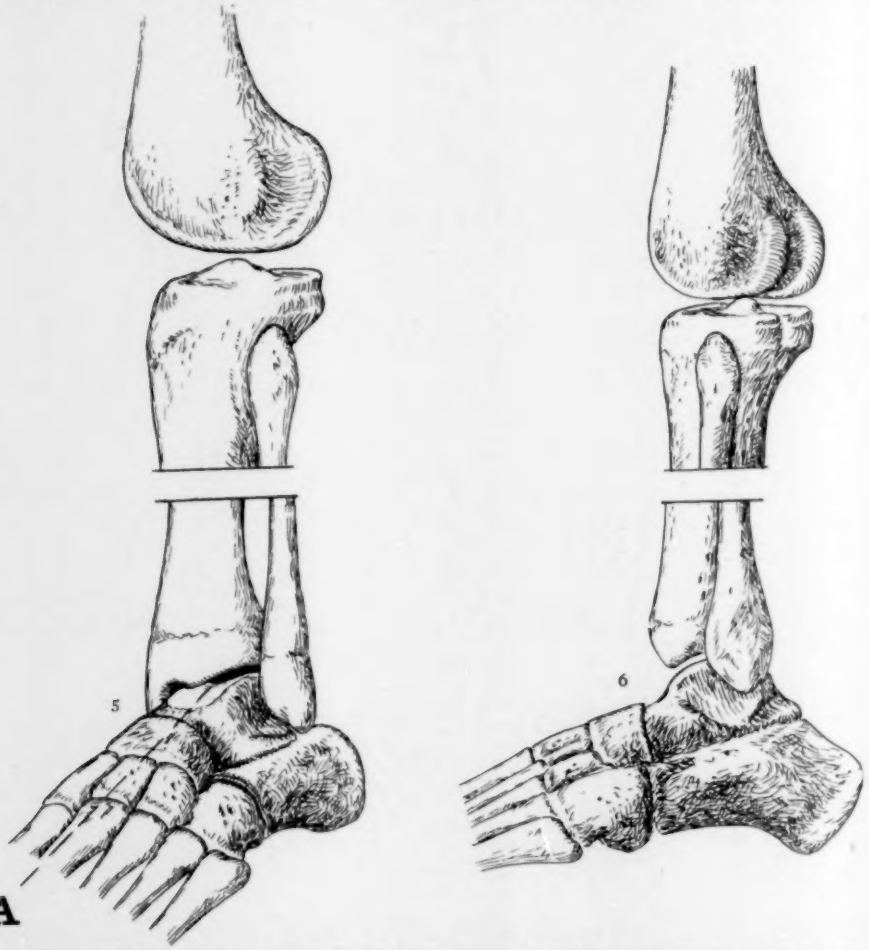
congenital club feet when the peroneals remain permanently weakened from overstretching.

2. Supination of the foot. The entire foot is supinated beneath the astragalus. The heel is inverted and the patient walks on the outer border of the foot.

3. Sub-astragalar inversion. The sub-astragalar joints are so designed that simple "hinged" motion does not occur through them in unison. As supination takes place, the foot revolves around a perpendicular axis which transfixes both the os-calcis and the astragalus in the region of the interosseous ligament. It roughly corresponds to the axis of the leg. As a result of this rotation, the fore-foot is drawn inward and the head of the astragalus

is directed toward the outer border of the inverted foot. The supinated foot is thus rotated simultaneously on two separate axes, the one, concerned with supination, extends obliquely antero-posterior, the other is perpendicular and pertains to sub-astragalar inversion.

The sub-astragalar inversion is usually masked by the eversion of the ankle-joint due to the external torsion of the lower leg. The two torsions



FIGS. 5 and 6.—5. Redressment of the Foot. Foot is placed in alignment with the ankle-joint (thus correcting the sub-astragalar inversion) and displaced backward bringing denuded surface of scaphoid in contact with the stump of the astragalar neck. Foot is everted in relation to knee owing to tibial torsion. 6. Operation completed. Foot viewed in profile and knee points inward. If tibial torsion impairs function it is corrected by osteotomy.

take place in opposite directions and thus compensate each other. As a result, the deformed foot appears to be in correct alignment with the limb, the fore-foot pointing forward in the same direction as the knee-joint.

4. Forward displacement of the foot beneath the astragalus. This movement takes place simultaneously with supination and sub-astragalar inversion. As a result, the heel is shortened. Developmental changes add to its shortening as the outer border of the foot becomes the chief point of weight-bearing.

5. Adduction of the fore-foot. Very little, if any, adduction is possible at the "Chopart" joint in the normal foot. In paralytic varus, the adduction is partly due to adaptive changes in the bones which form the calcaneo-cuboid joint. The anterior (articular) surface of the os-calcis which is ordinarily directed forward, faces inward and forward and the forward and outer corner of the bone becomes angular. Hoke refers to it as a "beak" (*Journal of Orthopedic Surgery*, vol. lx, 1911–1912). The convexity of the outer border of the adducted foot is chiefly due to these changes. For its correction, this "beak" should be trimmed off so that the anterior surface of the bone will face directly anterior.

The degree of adduction should be determined by inspecting the plantar surface of the foot and noting the convexity of its outer border. An exaggerated impression of the degree of adduction actually present is frequently formed when the dorsum of the foot and the front of the leg are viewed together. The inward deviation of the foot seen from this point of view is due to the sub-astragalar inversion in addition to the adduction which takes place at the "Chopart" joint.

Operative Considerations.—The importance of external torsion of the lower leg and the sub-astragalar inversion is not generally appreciated. If the deformed foot is "stabilized" and these two torsions are allowed to remain uncorrected, the ankle-joint will still be everted to both the foot and the leg. The motion of the joint will correspond to a line extending obliquely forward and outward through the foot and emerging somewhere on its outer border. Whenever the foot is plantar flexed, its outer border will be pitched downward in line with this motion. These movements continually throw the foot into supination (varus) and this state of affairs predisposes to the recurrence of the original deformity. Therefore, it is essential to correct the sub-astragalar inversion and place the foot in alignment with the ankle-joint. For similar reasons, the same principles apply to the correction of valgus deformities the mechanism of which is reversed. The "sub-astragalar eversion" present in the latter is equally as important.

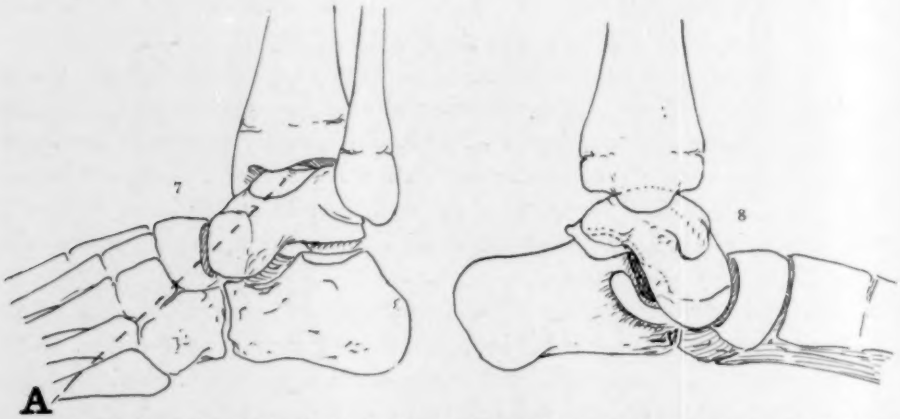
Supination, external torsion of the lower leg and sub-astragalar inversion are the three most important distortions of the deformity. Forward displacement of the foot beneath the astragalus is partly responsible for the shortening of the heel and should be corrected. Adduction of the fore-foot is less important as far as stability is concerned.

Description of the Operation.—1. Incision. The operative field is exposed by a curved incision on the outer side of the foot, similar to the one usually employed for astragalectomy. It is carried forward from beneath the tip of the external malleolus, its forward end curving upward to end on the dorsum of the foot immediately in front of the head of the astragalus. The outermost extensor tendons of the toes are brought into view and retracted upward out of the way. The incision is then carried down to the bones. The sub-astragalar joints are exposed and the head and neck of the astragalus

are freed from the overlying tissues. In the posterior end of the wound, the calcaneo-fibular ligament will be encountered. It should not be cut.

2. Resection of the sub-astragalar joints. The interosseous ligament is severed and a wedge of bone, base outward, is removed from between the os-calcis and the astragalus. The base of the wedge will ordinarily be one-fourth to one-half of an inch in thickness. It should be broad enough to correct the inversion of the heel.

3. Excision of the head of the astragalus. The excursion of the astraga-



FIGS. 7 and 8.—Comparison of skeletal changes of paralytic varus with paralytic valgus. Drawings made from X-ray tracings. Both deformities due to poliomyelitis occurring in infancy. 7. Paralytic varus, age nine, viewed from outer side. Foot is "in profile" while ankle is viewed obliquely due to external torsion of leg. "A" represents direction of ankle motion. Head of astragalus directed toward outer border of foot as a result of sub-astragalar inversion. Note shortening of heel due to forward displacement of foot beneath astragalus. Sub-astragalus joint horizontal. 8. Paralytic valgus, age ten, viewed from inner side. No torsion of lower leg. Note the long, prominent heel ("nigger heel") due to backward displacement of the foot (or stated reversely, forward displacement of astragalus). Astragalus head directed inward as a result of "sub-astragalar eversion." It is pitched downward and forward. Sub-astragalar joints nearly perpendicular. They should normally slope at angle of 40°.

lus in the tibio-fibular mortice is first carefully studied and the direction of the motion occurring at the ankle-joint determined. The astragalo-scaphoid joint is opened and the head is freed from the scaphoid. The neck of the astragalus is then cut across at right angles to the direction of the movements of the ankle-joint. The osteotome is entered on the outer side of the neck near its base (about five-eighths to three-fourths of an inch behind the outer margin of the head). It is directed obliquely inward and forward toward a point further forward on the inner side of the neck (closer to the inner margin of the head). The neck is thus cut obliquely across and the remaining portion of the neck will be somewhat longer on its inner aspect. The obliquity of the cut will depend on the eversion of the ankle. The cut end of the neck is shaped to conform to the concavity of the scaphoid which is likewise denuded of its joint cartilage.

4. Redressment of the foot. The foot is now freely movable beneath the astragalus. It is placed in alignment with the ankle-joint. This manœuvre brings the scaphoid in front of the astragalar neck and corrects the sub-astragalar inversion. The foot is then shifted backward beneath the astraga-

lus until the concave surface of the scaphoid is brought in firm contact with the stump of the astragalar neck which has been shaped to receive it. Supination, sub-astragalar inversion and forward displacement of the foot are thus corrected and the foot placed in correct alignment with the ankle-joint. The heel becomes more prominent and the external malleolus assumes a more normal position in relation to the foot.

If the soft parts resist correction, more bone is gouged from the sides of the "wedge gap" between the astragalus and os-calcis, allowing some of the bone particles to remain as grafts and fill in any irregularities of the cut surfaces. By thus increasing the breadth of the gap, these structures are further relaxed when the bony surfaces are opposed.

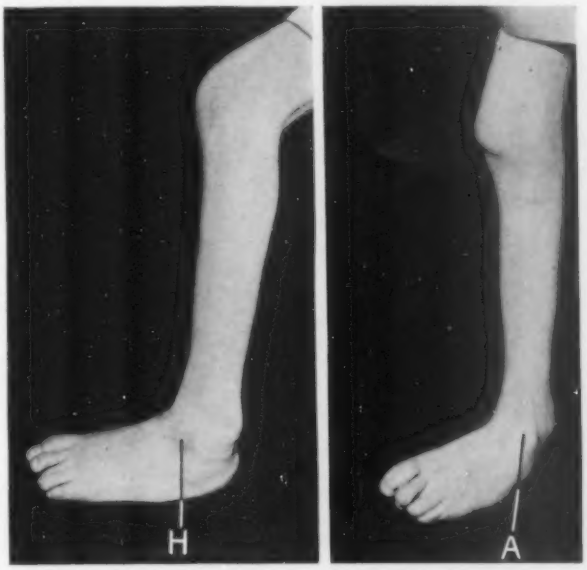


FIG. 9.—Paralytic talipes varus before operation. External torsion of the lower leg with eversion of the ankle-joint. Direction of ankle motion indicated by line "A." External malleolus "slumped" posteriorly. Foot is supinated and twisted inward beneath the everted ankle (sub-astragalar inversion), the forefoot pointing forward in same direction as the knee. As a result of the sub-astragalar inversion, head of astragalus (H) is partly uncovered and directed toward the outer border of the foot. Shortening of the heel due to forward displacement of the foot beneath the astragalus.

5. Arthrodesis of the calcaneo-cuboid joint. The adduction of the forefoot is determined by inspecting the sole of the foot. A gouge is then introduced into the joint which is accessible through the same incision. The articular cartilage is peeled off and the forward end of the os-calcis is "squared off" by crushing down its external forward "beak" sufficiently to correct the adduction, *i.e.*, until the outer border of the foot is no longer convex.

6. The wounds are closed in the usual manner and a well-moulded plaster case applied extending from the middle of the thigh to the toes. During its application, the backward displacement of the foot must be maintained, thus assuring firm contact between the denuded surface of the scaphoid and the cut end of the astragalar neck. This case is worn for five weeks. It is then replaced by a shorter case which extends from just below the knee to the toes and weight-bearing permitted. This second case is worn for about seven weeks.

At the conclusion of the post-operative treatment, the foot will be firmly stabilized in correct position. It will be in alignment with the ankle-joint. Owing to the external torsion of the lower leg, it will point somewhat out-

ward. If the external torsion responsible for the eversion of the foot is sufficient to impair function, it should be subsequently corrected by tibial osteotomy as Hoke has recommended. Moderate torsions are not disabling and do not demand correction.

These measures resemble the operation introduced several years ago by Hoke (*Journal of Orthopedic Surgery,* vol. xix, No. 10, 1921). In his operation, the "neck of the astragalus is cut through where it joins the body." The excised head is reshaped to fit its original bed, which has been altered by the redressment of the foot and then implanted into it as a graft. The technic is modified for the various deformities. An important advantage of his operation is the latitude it affords for the correction of the deformity. The operator "is able to correct the posterior foot deformity, etc.," and shift the foot backward as Whitman has pointed out as so essential in his astragalectomy." However, dependence on a free bone transplant is a weak link in the chain especially, since the graft is obliged to span a joint ("Chopart") which has not been completely resected.

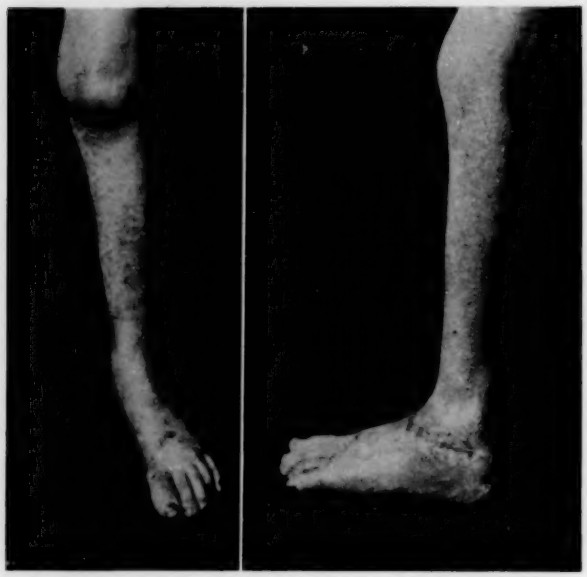


FIG. 10.—Paralytic talipes varus after operation. Note the improvement in the alignment and shape of the heel. Compare position of external malleolus to Fig. 9. Foot everted in relation to knee but not enough to interfere with function.

More recently, Smith and von Lackum (*Surgery, Gynæcology and Obstetrics,* vol. xl, No. 6, 1925) have described their method which is essentially a modification of Hoke's operation for calcaneus minus the bone transplant. In addition, they resect the calcaneo-cuboid joints. They cut the neck of the astragalus across transversely and place the foot in alignment with the leg. No mention is made of the ankle-joint. To place the foot thus in the presence of tibial torsion for the correction of varus deformities, disregards the sub-astragalar inversion. Their operation would seem to be more suitable for the correction of calcaneus and cavus.

In the operation which I have described, the foot is placed in alignment with the ankle-joint. The head of the astragalus is excised by cutting the neck obliquely across so that when the sub-astragalar inversion is corrected and the foot placed in alignment with the ankle, the cut surface of the neck will then be transverse in relation to the corrected foot. This operation is not suitable for the other forms of talipes.

CONCLUSIONS

1. It is essential to correct the sub-astragalar inversion and place the foot in alignment with the ankle-joint.

2. In the presence of external torsion of the lower leg, the foot will point outward when placed in alignment with the everted ankle. If the torsion is sufficient to impair function, it should be subsequently corrected by tibial osteotomy, as Hoke has recommended.

3. Sub-astragalar torsions are important in lateral deformities of the feet. In varus, sub-astragalar inversion takes place with supination. Sub-astragalar eversion occurs in valgus. These torsions are corrected when the foot is placed in alignment with the ankle.

PARAFFINOMA OF THE KNEE

By Samuel Kleinberg, M.D.
of New York, N. Y.

Paraffin tumors are seen very frequently in the face, neck and even in the hands as a result of deliberate injection for cosmetic purposes. In an intensive search of the literature, however, I have not come across a case like the one now reported, in which a successful attempt was made to produce a swelling and inflammation of a knee-joint to avoid military service. This case is interesting also because we had the opportunity of studying the tumor tissue microscopically eight years after the injection.

Case Report.—G. M., twenty-seven years old, injected eight years ago about an ounce of liquid paraffin at various points into the subcutaneous tissues of the front of his left knee. In order to increase the irritative effect of the oil he beat his knee with a board. There resulted a disabling, painful but afebrile swelling of the knee. He was admitted to a hospital where he was treated for "synovitis" for three months. The inflammation gradually subsided and he had no further trouble for three years. Since then he has had attacks of painful swelling of the knee about once a year. These attacks appeared without any apparent cause, and all except the present one lasted only a week or two. Three months ago the knee became swollen and the discomfort and disability have persisted.

Examination.—The patient was in good general condition. He walked without assistance, but with a left limp. The left knee (Fig. 1) was enlarged because of several tumors. There was an oblong mass about two inches wide and four inches long on the outer side of the knee lateral to the patella, beginning on a level with its upper border and extending downward. It was semi-solid in consistency, irregular in outline, but fairly well demarcated. It was slightly movable on the deep tissues, but was attached to the skin. There was no redness, extra heat or tenderness. Several similar masses were present on the inner side of the knee. The motions in the knee-joint were practically unrestricted. Several glands in the groin were slightly enlarged. Röntgen-ray pictures were entirely negative except for irregular dense shadows in the subcutaneous tissue. As the skin was adherent to the tumors, I was certain that I would have to remove a large part of it when the masses were excised. Hence it was decided to perform the operation in two stages in order to be able to mobilize the skin for closure of the gap created at the time of operation.

First Operation, January 21, 1926.—A five inch incision was made over the antero-external aspect of the left knee. Immediately below the skin and attached to it a very hard mass was found. It was so thoroughly adherent to the skin that a second incision was made through the skin joining the original incision above and below, and about three-fourths of an inch away from it at the middle. The tumor with the attached oval section of skin was separated from the surrounding tissues. It was found to infiltrate the superficial fascia but was quite free from the deep fascia. It was removed *en masse.* There was an abundant blood supply at the periphery of the tumor. There were numerous small, very hard tumors embedded in the deep layers of the skin and in the subcutaneous fascia in the vicinity of the main tumor; these were excised. The wound was closed with several layers of sutures and healed by primary union.

Second Operation, February 15, 1926.—An oval incision was made on the antero-

internal surface of the left knee. Several tumors were located and all of them, including some of the overlying skin, were removed. There location in the subcutaneous tissues, consistency, attachment and distribution were similar to those described in the first operation. The wound was closed with a layer of catgut sutures for the subcutaneous tissues and silk for the skin, and healed by primary union.

The gross appearance of all of the tumors was the same. Each mass was hard and on section cut like dense fibrous tissue or cartilage. The cut surface was white

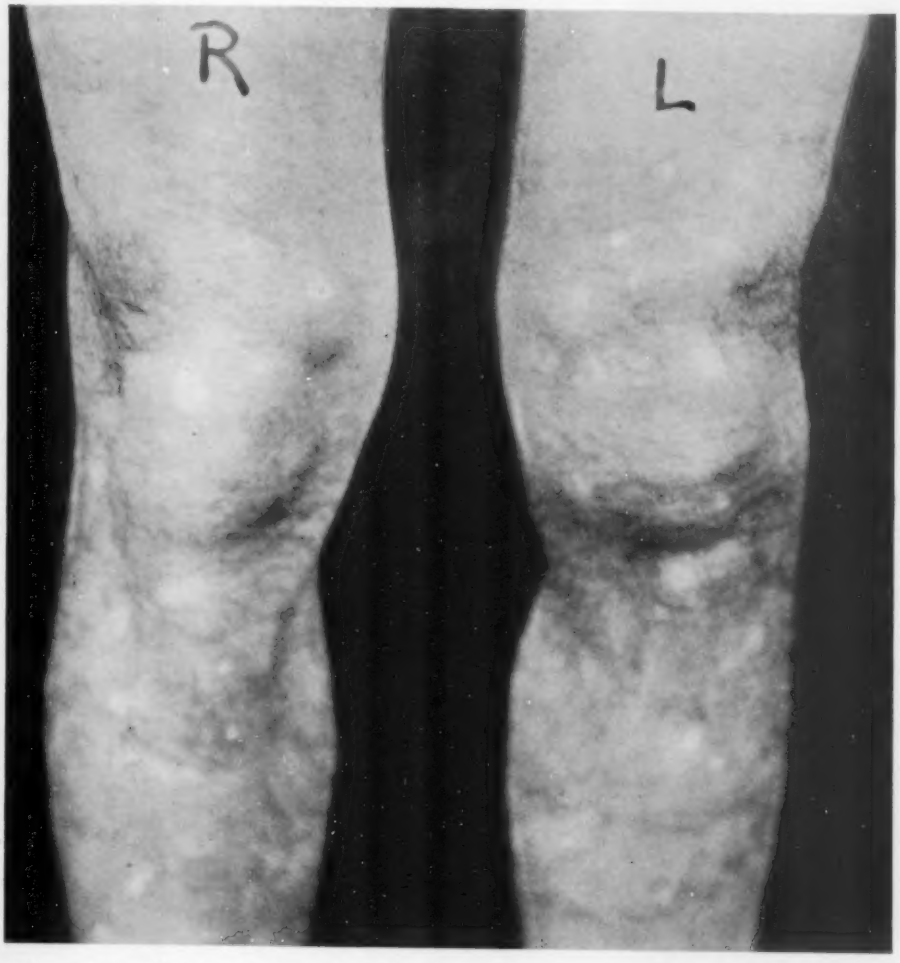


FIG. 1.—Front view of both knees. There is an evident enlargement of the left knee (L) with nodular thickening above the patella and on the lateral aspects of the knee.

except for a few streaks of yellow. In contrast to the abundant blood supply about the tumor there were very few blood-vessels in the tumor substance. There was no evidence of either fluid or solid paraffin.

Microscopical sections (Fig. 2) from the tumors are identical. They show that the tumors consist of very dense fibrous connective tissues, numerous areas of small cell infiltration and many fat cells. There is no limiting membrane. There are no giant cells such as have been found and described by many authors reporting paraffin tumors about the face. There are, however, a very large number of blood-vessels undergoing necrosis, evidently the result of compression and strangulation by the cicatricial tissue.

The areas of small cell infiltration are the result of the exudative inflammatory reactions during the "attacks" of painful swelling.

In this case, seen eight years after the injection, there was no evidence of any paraffin which had evidently been completely absorbed and replaced by dense fibrous connective tissue. Periodically there were inflammatory reactions resulting in swelling and pain of the knee. It is difficult to account for the exacerbations unless it be that the last vestige of oil disappeared during

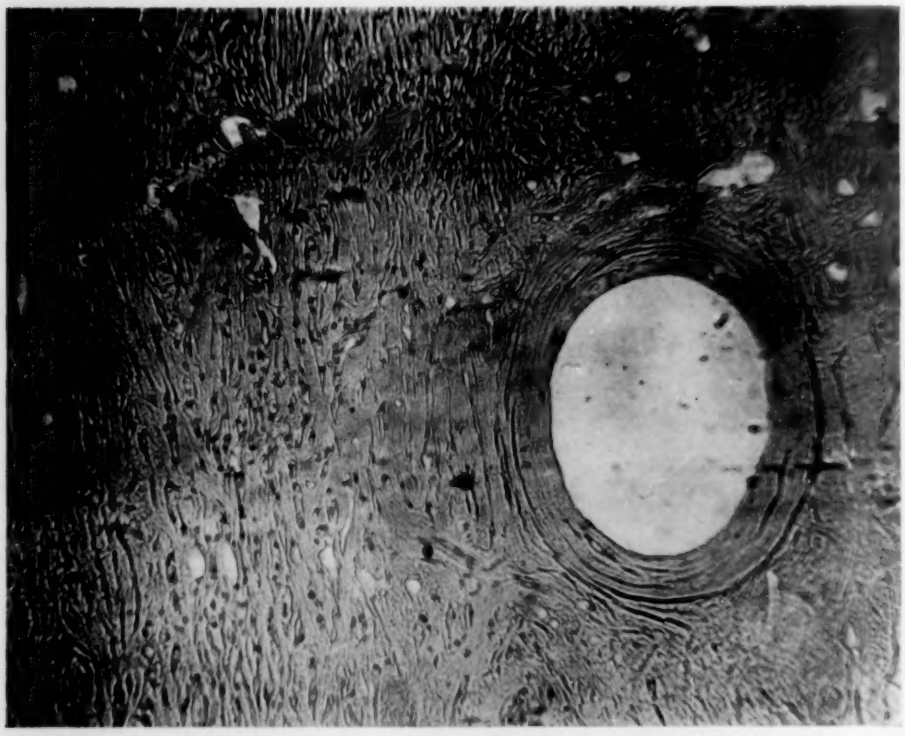


FIG. 2.—Microphotograph of paraffinoma. Low power. There is a very dense fibrous connective tissue with small cell infiltration. Note the blood-vessel undergoing necrosis, probably as a result of contraction and strangulation by the connective tissue. There are no foreign body giant cells and no encapsulated areas of paraffin.

the last inflammation. My case would tend to prove that with a low melting point paraffin is split up and is gradually absorbed. Contrariwise, paraffin with a high melting point remains practically unchanged in the body tissues. It is interesting to note, too, that whereas the literature abounds in many case reports recording the movement of the paraffin to points at some distance from the original site of injection, in my case there was no migration, although the subcutaneous tissues about the knee are rather loose, and one would expect, since the knee is in the vertical position so much of the day, that the force of gravity would cause the paraffin to move about, especially downward.

BRIEF COMMUNICATIONS

BRACKETT'S OPERATION FOR HIP FRACTURES

DOCTOR HENDERSON, in his interesting paper on the operative treatment of ununited fracture at the hip at the Mayo Clinic, has evidently failed to verify his references. What he describes, or at least illustrates, as a Brackett operation, is a transplantation of the trochanter to the shaft, the essential feature of the reconstruction operation, and apposition of the base of the neck to the head.

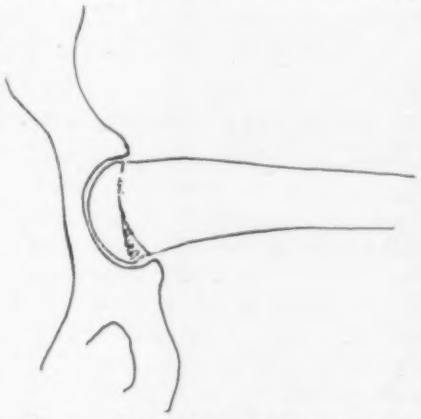


FIG. 1.—Brackett's operation according to Doctor Henderson shows a transplanted trochanter and apposition of the neck and head.

Brackett's operation (*Bost. Med. and Surg. J.*, 1917, vol. clxxvii) was entirely different in design. He removed the muscular attachments, together with a section of underlying bone from the upper extremity of the trochanter and applied the upper and inner part of this trochanteric surface to the freshened head, the muscular attachments being placed about the junction of the two. It was assumed that the nutrition of the trochanteric extremity of the femur would assure a better opportunity for repair than the atrophied remains of the neck. A comparison between Brackett's illustration and that representing Doctor Henderson's conception of the Brackett operation will show clearly the essential distinction between the two.

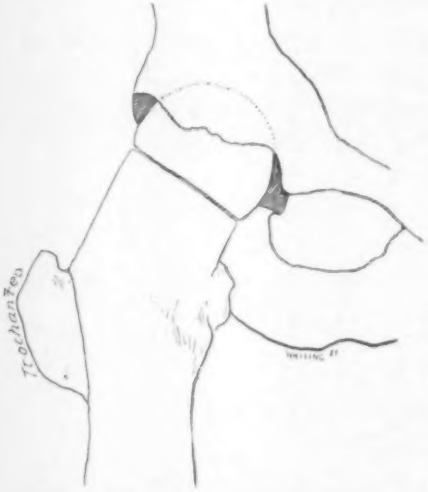


FIG. 2.—Brackett's operation according to Doctor Henderson shows a transplanted trochanter and apposition of the neck and head.

As regards bone pegging—I am convinced that the field for this operation should be, practically speaking, restricted to cases in which primary treatment has been ineffective, and in which, therefore, the capacity of the tissues for repair has not been tested. For, if the abduction treatment has been properly applied, in the sense that the apposition of the fragments has been verified by X-ray examination, failure of union is usually accompanied by extensive disintegration of the neck. In such cases the prospect of repair after bone grafting is so uncertain

that one would be hardly justified in undertaking an operation which, even if successful, offers so slight an advantage from the functional standpoint over the reconstruction operation.

ROYAL WHITMAN, M.D.,
New York, N. Y.

INGUINO-PERINEAL HERNIA COMPLICATED WITH ECTOPIA TESTIS

From a brief search of text-book and journal literature and inquiry among colleagues, it would seem that inguino-perineal hernia is quite a rare condition; because of this fact and because of the interesting conditions of embryological and anatomical interest presented, this case is reported:

M., male, age forty-two, complained that all his life his right testicle had been misplaced and more or less fixed in the right side of the perineum and that for sixteen or eighteen months he had noticed a hernia on the same side.

Examination showed a large right-sided inguinal hernia, protruding through the inguinal canal, not entering the scrotum but entering for a short distance the upper or anterior part of a loose baggy fold formed by the right half of the perineum. The post-central part of this baggy fold contained the right testicle which was fixed rather firmly. Operation showed a large thick hernial sac with the processus vaginalis patent to the anterior part of the perineum; firmly closed beyond it, ending in a thick, broad mesorchium containing the cord and later opening up to form a roomy tunica vaginalis which contained the slightly undersized testicle, firmly fixed to its perineal site.

The processus vaginalis extended about an inch further posteriorly than the testicle or epididymis. The posterior limit of the vaginal process was almost opposite the anus. The right half of the scrotum was undeveloped and, of course, contained no peritoneal structure.

Inguino-perineal hernias follow the inguinal canal and enter or threaten to enter the perineum instead of the scrotum. They must not be confused with true perineal hernias which protrude through the pelvis.

The condition is a congenital one and is a result of the vaginal process of peritoneum being pulled down by the development of that portion of the gubernaculum testis which is attached in the perineum—other attachments of the gubernaculum are found in the regions of the saphenous opening, the anterior superior spine of the ilium and the pubes and may be followed by displacements of the testicle in these areas.

WALTER D. WISE, M.D.,
Baltimore, Md.

PERFORATED GANGRENOUS APPENDIX IN INGUINAL HERNIA

A woman, aged forty-two, married, presented herself for treatment at the Franklin Square Hospital of Baltimore. Two weeks prior to admission she had noticed a small, tender swelling in her right groin. This mass had gradually increased in size and had become painful. No history of a mass of any kind previously. Personal history—other than a creamy vaginal discharge and dysmenorrhœa—revealed nothing of import. There was no nausea or vomiting and no disturbance of bowel or urinary elimination.

The right inguinal region was markedly swollen, red and extremely tender. In

appearance it suggested localization of an infective process. The mass was soft and fluctuant, every evidence of free pus. Peculiarly the swelling seemed to involve the course of the inguinal canal. There was no enlargement of the left inguinal region. The white blood count was 31,200, polymorphneutrophiles 77 per cent. Temperature on admission 101.8, pulse 95.

The woman was sent to the operating room where an incision similar to that done for inguinal hernia was made. A half pint of creamy yellow material flushed out of the wound, with an odor typical of colon bacilli. On exploring the region, the finger encountered an olive-shaped mass which, when palpated, resembled an infected inguinal gland. Enucleation of this mass from its bed revealed a gangrenous, perforated appendix, constricted, as it were, by peritoneum at its base. The appendix was carefully drawn out of the wound, ligated, cauterized at its normal base and the stump allowed to fall back in abdominal cavity. A cigarette drain was inserted into the abdomen. The internal oblique muscle was sutured to the shelf of Poupart's ligament, allowing room for free drainage. The fascia was sutured, skin wound closed with one drain protruding.

Patient's temperature the day following operation had dropped to 100 degrees and then gradually approached the normal. The drainage tube was removed on the third day. The wound healed by granulation; there were no untoward effects or complications. The patient was seen in the out-patient department at weekly intervals for the period of a month. Was last seen three months after primary operation. There were no complications of any kind, good granulation of wound, no evidence of a hernial mass.

Louis Antupit, M.D.,
Baltimore, Md.

ULCER OF THE JEJUNUM

Ulcer of the jejunum close to the duodeno-jejunal flexure and behind the stomach is apparently a very infrequent occurrence. Symmers [1] reports that any pathology in the oral end of the jejunum is very uncommon and that ulcer is exceedingly rare. No case appears on the records at Bellevue Hospital.

Jejunal ulcers are found about the stoma after gastro-enterostomy. Ashcroft [2] describes three perforated jejunal ulcers, two in one patient, after posterior gastro-jejunostomies. These occurred in the efferent loops. Under experimental conditions, one finds organic disturbance in the afferent and efferent intestinal loops about the gastro-enterostomy that vary from simple congestion of the mucosa to frank ulceration. During the first two weeks there are congestion, swelling, œdema, and superficial necrosis of the lower portion of the duodenum and jejunum about the gastric stoma. These changes were not observed farther than twenty centimetres aboral of the stomach. They were thought to be essentially due to contact between the alkaline intestine and the acid digestive juices from the stomach. In later animals in a small percentage of instances marginal chronic ulcers were found. The suddenness of the exposure of the jejunum to the irritating stomach contents is considered largely accountable for the acute inflammatory swelling of the mucosa and submucosa, for with time the great proportion of these gastro-enteromized animals became free from these symptoms. This experience in experimentally controlled animals corresponds with the symptoms we meet with in some of our patients, after gastro-enterostomy.

The following case had not had gastro-enterostomy but had had two acute gastric perforations; one in 1917, one in 1922, both of which had healed completely after simple inversion before the present acute perforated ulcer in the post-gastric jejunum occurred.

Patient is J. V. D., aged thirty-three years, a cook by occupation, a native of

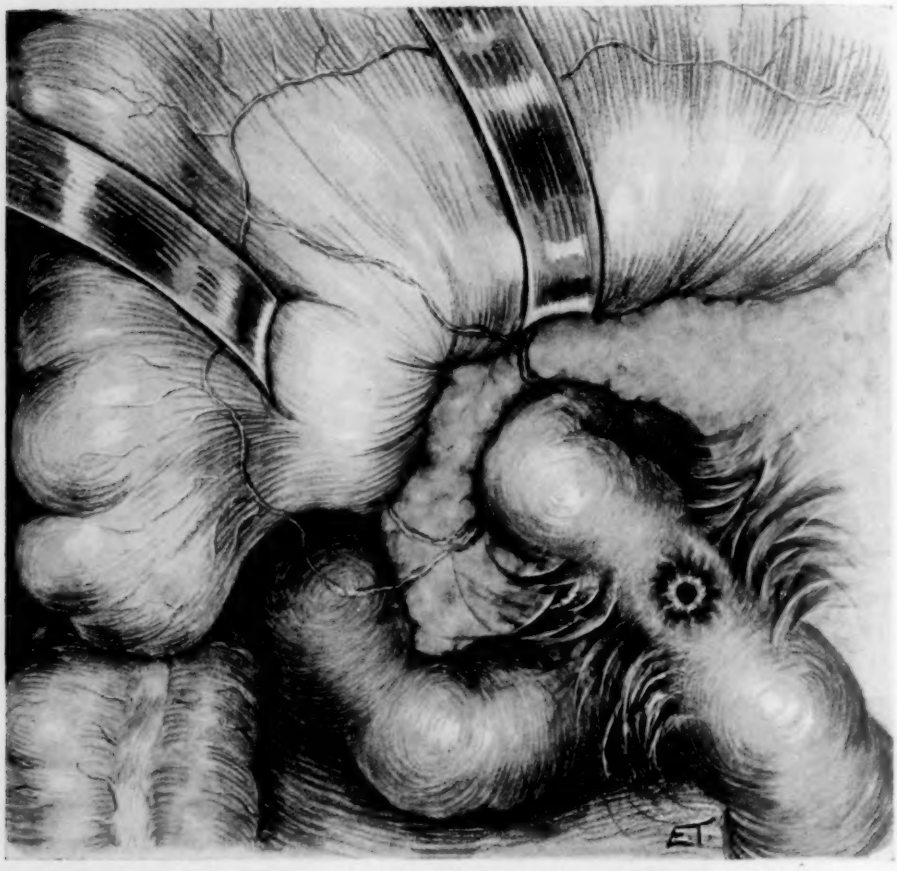


FIG. 1.—Ulcer of the oval jejunum behind the stomach. Note stomach and colon raised. Duodenum emphasized to show the relation of the ulcer about two fingers' breadth below the duodeno-jejunal flexure and ulcer inadequately protected by adhesions.

U. S. A., who was admitted to the Jamaica Hospital, May 21, 1926. Father died at thirty-four and mother at forty-four of causes unknown. He has three brothers and four sisters living and well. He denies venereal disease; is a father of two healthy children. Reports he has been well until the first attack of abdominal pain and operation at Bellevue Hospital in 1917 for acute perforated gastric ulcer. Following this operation he was well for a few months and then experienced recurrent pains in the epigastrium about three and a half hours after meals until 1922, when he was again operated at Bellevue for another perforated gastric ulcer. He was symptom-free after this second operation until the sudden attack of severe pain at the umbilicus, six hours before his third operation at Jamaica Hospital, May 21, 1926.

The findings were: large post-operative epigastric hernia, generalized peritonitis, peritoneum flooded with bile and gastric contents; stomach, transverse colon, and omentum

adherent to scar, healed ulcer in anterior wall of pylorus, stomach distended, no evidence of gas under pressure or of abnormal gastric emptying sound observed upon entering the peritoneum, perforated and moderately indurated ulcer, 1 cm. in diameter, in the jejunum, anterior wall and two fingers' breadth below the flexure (Fig. 1). The induration extended into the adjoining peritoneum about which there were some adhesions. These adhesions were not sufficient to protect the ulcer, for while the perforation was under observation the outer coat of a green pea was seen to emerge from the opening. The operative treatment consisted in inverting the ulcer with three mattress sutures of Pagenstecher, posterior gastrojejunostomy, and drainage of lesser sac, Morrison's space, and pelvis (the latter through a stab wound above the pubis). The hernia was repaired as well as possible in closing the abdomen. The patient was treated for shock and peritonitis immediately following the operation. He was discharged from the hospital after a remarkably smooth convalescence of three weeks. At the return-clinic up to August 20, he is reported symptom-free, increased in weight, and back at work.

REFERENCES

[1] Symmers, Douglas: Personal Communication.
[2] Ashcroft: Brit. Med. Jour., vol. i, pp. 515–554, Mar. 20, 1926.

W. Howard Barber, M.D.,
New York, N. Y.

BOOK REVIEW

SCOLIOSIS—ROTARY LATERAL CURVATURE OF THE SPINE. By SAMUEL KLEINBERG, M.D. Paul B. Hoeber, Inc., New York, 1926, octavo, pp. 311.

THIS single volume is a concise and readily comprehended review of the experience of the author concerning scoliosis during the past few decades. The work comprises fourteen chapters which are titled as follows: 1. Introduction. 2. Anatomical and Physiological Considerations. 3. Classification and Pathology. 4. Etiology. 5. History and Methods of Examination. 6. Clinical Records. 7. General Symptoms. 8. Prophylaxis. 9. Treatment. 10. Gymnastic Exercises. 11. Forcible Correction. 12. The Operative Treatment of Scoliosis. 13. Treatment of Special Types of Scoliosis. 14. Prognosis. It deviates from the usual text-book in two essential manners; in the first place the apparatus described herein is far less cumbersome and complicated than that formerly used. It gives briefly the various methods of treating different types of scoliosis by mechanical devices and exercises at the command of the orthopædic surgeon in his office and the general hospital. In the second place the terminology and classification has been greatly simplified so that it is decidedly easier to follow the descriptive matter than in works in which the classification is so much more complicated. There is intentionally an absence of descriptive mechanotherapy due to the fact that the apparatus used by the author is so simple that there seems to be no excuse for using large, cumbersome machines for the correction of this condition.

While this book is evidently written essentially for the orthopædic surgeon, its presentation is so simple, straightforward and logical that it is easily followed by general practitioner in spite of the fact that subject is a highly specialized one. Chapters on anatomical physiological considerations, prophylaxis, gymnastic exercises and forcible corrections are so complete yet concise, so complicated but so simply presented that they are indeed noteworthy.

In the author's preface he frankly states that of course quotations from the works of other orthopædic surgeons have been freely employed. Nevertheless, the great bulk of material, particularly that pertinent to the clinical pathology, etiology, classification and treatment, is the result of his own detailed and intensive study and observations in this field of work. The reviewer feels that if more highly technical considerations of the various other specialty conditions and diseases could be as simply and as concretely expressed as in this work they would find an interested and eager group of readers among the medical profession. MERRILL N. FOOTE.

To Contributors and Subscribers:

All contributions for Publication, Books for Review, and Exchanges should be sent to the Editorial Office, 145 Gates Ave., Brooklyn, N. Y.

Remittances for Subscriptions and Advertising and all business communications should be addressed to the

ANNALS of SURGERY
227-231 So. 6th Street
Philadelphia, Penna.